L14 ANSWER 22 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AB Behcet's syndrome is a multisystem vasculitis of unknown aetiology. Eye involvement, the main cause of morbidity, can lead to blindness in 20% of those affected. Other lesions, ranging from **aphthous** and genital ulceration to sometimes fatal central nervous system involvement, also cause considerable morbidity and, as we have become more recently aware, mortality. The syndrome runs a course of exacerbations and remissions, and usually abates in intensity with the passage of time. Young adult males have the worst prognosis. The main aim of treatment is to prevent irreversible organ damage during-the early, active, phase of the disease. Immunosuppression remains the mainstay of therapy. Azathioprine is able to suppress most of the manifestations of the syndrome. **Cyclosporin** has a considerably more rapid onset of action, and is particularly useful in the treatment of uveitis. However, the disease usyally flares on cessation of **cyclosporin** treatment. Neither azathioprine nor **cyclosporin** is always effective, and there are patients who continue to do badly even with their combined use. **Thalidomide** is useful in severe oral ulceration and colchicine in erythema nodosum associated with Behcet's syndrome. There is no established remedy for the central nervous system and thrombotic complications of Behcet's syndrome.

AN 95056456 EMBASE

DN 1995056456

TI Behcet's syndrome: How should we treat it?.

AU Yazici H.; Yurdakul S.; Hamuryudan V.

CS Division of Rheumatology, Dept. Med. Cerrahpasa Med. Faculty, Safa Sok 17/4,Kadikoy, 81310 Istanbul, Turkey

SO Clinical Immunotherapeutics, (1995) 3/2 (102-107).
ISSN: 1172-7039 CODEN: CIMMEA

CY New Zealand

DT Journal; General Review

FS 006 Internal Medicine
011 Otorhinolaryngology
012 Ophthalmology
025 Hematology
026 Immunology, Serology and Transplantation
031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles

LA English

SL English

SO Clinical Immunotherapeutics, (1995) 3/2 (102-107).
ISSN: 1172-7039 CODEN: CIMMEA

L14 ANSWER 11 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB The cause of recurrent **aphthous** ulcers (RAU), the lesions of recurrent **aphthous** stomatitis, is incompletely understood but appears to involve immune system dysfunction. Treatment options include no treatment, treatment of associated systemic diseases or conditions (eg, celiac sprue, vitamin deficiencies), systemic medications, topical medications, conversion of the **aphthous** ulcer to a wound, and palliative treatments. The most effective treatments (systemic or topical corticosteroids, **thalidomide**) involve agents that suppress or modulate immune system function. In general, topical agents are preferred because they have fewer associated side effects; however, inability to obtain adequate contact time may limit their effectiveness. Adjunct pain control is sometimes necessary ether with pain medications or with adherent agents that coat the ulcers.
AN 1998004823 EMBASE
TI Topical and systemic therapy for recurrent **aphthous** stomatitis.
AU MacPhail L.
CS Dr. L. MacPhail, UCSF, Department of Stomatology, Box 0422, 513 Parnassus, San Francisco, CA 94143-0422, United States
SO Seminars in Cutaneous Medicine and Surgery, (1997) 16/4 (301-307).
Refs: 78
ISSN: 1085-5629 CODEN: SCMSFR
CY United States
DT Journal; Conference Article
FS 011 Otorhinolaryngology
037 Drug Literature Index
LA English
SL English
TI Topical and systemic therapy for recurrent **aphthous** stomatitis.
SO Seminars in Cutaneous Medicine and Surgery, (1997) 16/4 (301-307).
Refs: 78
ISSN: 1085-5629 CODEN: SCMSFR

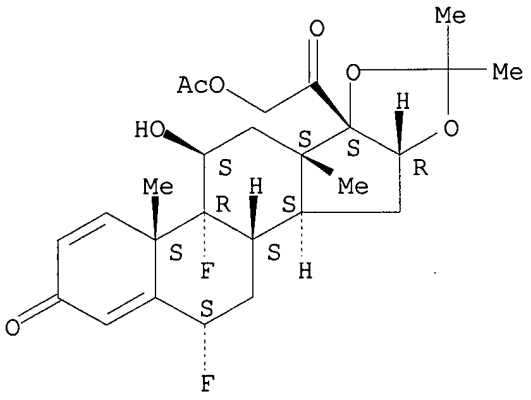
Calculated physical property data is now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

=> s fluocinonide/cn
L1 1 FLUOCINONIDE/CN

=> d

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2002 ACS
RN 356-12-7 REGISTRY
CN Pregna-1,4-diene-3,20-dione,
21-(acetyloxy)-6,9-difluoro-11-hydroxy-16,17-
[(1-methylethylidene)bis(oxy)]-, (6.alpha.,11.beta.,16.alpha.)- (9CI)
(CA
INDEX NAME)
OTHER CA INDEX NAMES:
CN 2H-Naphth[2',1':4,5]indeno[1,2-d][1,3]dioxole,
pregna-1,4-diene-3,20-dione
deriv.
CN Pregna-1,4-diene-3,20-dione,
6.alpha.,9-difluoro-11.beta.,16.alpha.,17,21-
tetrahydroxy-, cyclic 16,17-acetal with acetone, 21-acetate (7CI, 8CI)
OTHER NAMES:
CN Flucinar
CN Fluocinolide
CN Fluocinolide acetate
CN Fluocinolone acetonide 21-acetate
CN Fluocinolone acetonide acetate
CN **Fluocinonide**
CN Lidex
CN Lidex E
CN Metosyn
CN Topsym
FS STEREOSEARCH
MF C26 H32 F2 O7
CI COM
LC STN Files: ADISNEWS, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DIOGENES, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, NIOSHTIC, PHARMASEARCH, PROMT, RTECS*, SPECINFO, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

280 REFERENCES IN FILE CA (1967 TO DATE)
4 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
280 REFERENCES IN FILE CAPLUS (1967 TO DATE)
7 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> s PTX/cn
L2 1 PTX/CN

=> d

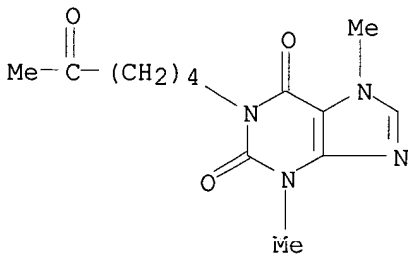
L2 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2002 ACS
RN 6493-05-6 REGISTRY
CN 1H-Purine-2,6-dione, 3,7-dihydro-3,7-dimethyl-1-(5-oxohexyl)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Theobromine, 1-(5-oxohexyl)- (7CI, 8CI)
OTHER NAMES:
CN 1-(5-Oxohexyl)-3,7-dimethylxanthine
CN 1-(5-Oxohexyl)theobromine
CN 3,7-Dihydro-3,7-dimethyl-1-(5-oxohexyl)-1H-purine-2,6-dione
CN 3,7-Dimethyl-1-(5-oxohexyl)-1H,3H-purin-2,6-dione
CN 3,7-Dimethyl-1-(5-oxohexyl)xanthine
CN Agapurin Retard
CN BL 191
CN Dimethyloxohexylxanthine
CN Oxpentifylline
CN Pentoxifyllin
CN Pentoxifylline
CN Pentoxiphyllin
CN Pentoxiphylline
CN Pentoxyfilline
CN Pentoxyphyllin
CN **PTX**
CN Torental
CN Trental
FS 3D CONCORD
MF C13 H18 N4 O3
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS,

CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DIOGENES, DRUGNL, DRUGPAT, DRUGU, DRUGUPDATES, EMBASE, IPA, MEDLINE, MRCK*, NIOSHTIC, PHAR, PHARMASEARCH, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL, VETU

(*File contains numerically searchable property data)

Other Sources: EINECS**, WHO

(**Enter CHEMLIST File for up-to-date regulatory information)



514/248

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1797 REFERENCES IN FILE CA (1967 TO DATE)
22 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1800 REFERENCES IN FILE CAPLUS (1967 TO DATE)
2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

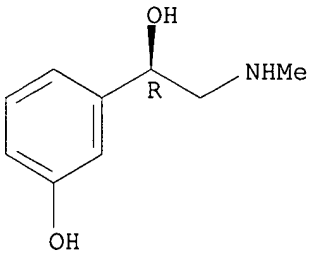
```
=> s decadron/cn
L3            2 DECADRON/CN

=> d

L3   ANSWER 1 OF 2  REGISTRY  COPYRIGHT 2002 ACS
RN   61-76-7  REGISTRY
CN   Benzenemethanol, 3-hydroxy-.alpha.-[(methylamino)methyl]-, hydrochloride,
     (.alpha.R)- (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Benzenemethanol, 3-hydroxy-.alpha.-[(methylamino)methyl]-, hydrochloride,
     (R)-
CN   Benzyl alcohol, m-hydroxy-.alpha.-[(methylamino)methyl]-, hydrochloride,
     (-)- (8CI)
OTHER NAMES:
CN   (-)-.alpha.-Hydroxy-.beta.-(methylamino)ethyl-.alpha.-(3-
     hydroxybenzene)hydrochloride
CN   (-)-Phenylephrine hydrochloride
CN   (R)-Phenylephrine hydrochloride
CN   Adrianol
CN   Almefrin
CN   Decadron
CN   Isophrin hydrochloride
CN   l-.alpha.-Hydroxy-.beta.-methylamino-3-hydroxy-1-ethylbenzene
     hydrochloride
CN   l-1-(m-Hydroxyphenyl)-2-methylaminoethanol hydrochloride
CN   l-m-Hydroxy-.alpha.-[(methylamino)methyl]benzyl alcohol hydrochloride
CN   l-Phenylephrine hydrochloride
CN   Levophenylephrine hydrochloride
CN   Lexatol
CN   Meta-Sympatol
```

CN Meta-Synephrine hydrochloride
CN Metaoxedrine chloride
CN Metaoxedrine hydrochloride
CN Mydfrin
CN Neo-Synephrine hydrochloride
CN Neo-Synesin 1
CN Neophryn
CN Oftalfrine
CN Phenylephrine hydrochloride
CN Prefrin
CN R-(-)-m-Synephrine hydrochloride
CN Sucraphen
CN Synasal
FS STEREOSEARCH
DR 644-22-4, 827-62-3, 50741-76-9
MF C9 H13 N O2 . Cl H
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS,
      BIOTECHNO, CA, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN,
      CSCHEM, DIOGENES, EMBASE, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MRCK*,
      MSDS-OHS, NIOSHTIC, PROMT, RTECS*, SYNTHLINE, TOXCENTER, USAN, USPATFULL
        (*File contains numerically searchable property data)
    Other Sources: DSL**, EINECS**, TSCA**
        (**Enter CHEMLIST File for up-to-date regulatory information)
CRN (59-42-7)

Absolute stereochemistry.



● HCl

784 REFERENCES IN FILE CA (1967 TO DATE)
  2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
784 REFERENCES IN FILE CAPLUS (1967 TO DATE)
  4 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

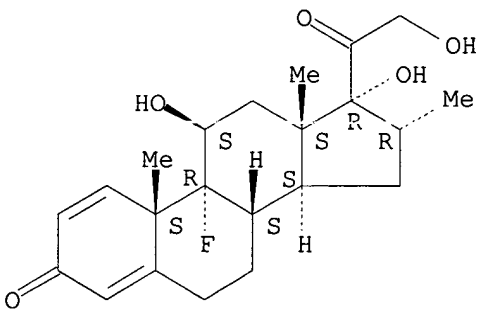
=> d 2

L3 ANSWER 2 OF 2 REGISTRY COPYRIGHT 2002 ACS
RN 50-02-2 REGISTRY
CN Pregna-1,4-diene-3,20-dione, 9-fluoro-11,17,21-trihydroxy-16-methyl-, (11.beta.,16.alpha.)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1-Dehydro-16.alpha.-methyl-9.alpha.-fluorohydrocortisone
CN 16.alpha.-Methyl-9.alpha.-fluoro-.DELTA.1-hydrocortisone

CN 16.alpha.-Methyl-9.alpha.-fluoro-1,4-pregnadiene-11.beta.,17.alpha.,21-triol-3,20-dione
CN 16.alpha.-Methyl-9.alpha.-fluoro-11.beta.,17.alpha.,21-trihydroxypregna-1,4-diene-3,20-dione
CN 16.alpha.-Methyl-9.alpha.-fluoroprednisolone
CN 9-Fluoro-11.beta.,17,21-trihydroxy-16.alpha.-methylpregna-1,4-diene-3,20-dione
CN 9.alpha.-Fluoro-11.beta.,17.alpha.,21-trihydroxy-16.alpha.-methyl-1,4-pregnadiene-3,20-dione
CN 9.alpha.-Fluoro-16.alpha.-methyl-1,4-pregnadiene-11.beta.,17.alpha.,21-triol-3,20-dione
CN 9.alpha.-Fluoro-16.alpha.-methyl-11.beta.,17,21-trihydroxypregna-1,4-diene-3,20-dione
CN 9.alpha.-Fluoro-16.alpha.-methylprednisolone
CN Aphtasolon
CN Aphthasolone
CN Azium
CN Calonat
CN Corsone
CN Decaderm
CN **Decadron**
CN Decasone
CN Dectancyl
CN Dekacort
CN Deltafluorene
CN Dergramin
CN Deronil
CN Desadrene
CN Desameton
CN Dexa-Cortidelt
CN Dexa-Cortisyl
CN Dexa-Scheroson
CN Dexacort
CN Dexadeltone
CN Dexalona
CN Dexaltin
CN Dexameth
CN Dexamethasone
CN Dexamethasone alcohol
CN Dexapolcort
CN Dexaprol
CN Dexason
CN Dexasone
CN Dexonium
CN Dextelan
CN Fluorocort
CN Gammacorten
CN Hexadecadrol
CN Hexadrol
CN HL-Dex
CN Luxazone
CN Maxidex
CN Millicorten
CN MK 125
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
FS STEREOSEARCH
DR 8054-59-9, 137098-19-2
MF C22 H29 F O5

CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*,
BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS,
CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU,
DIOGENES, DRUGU, EMBASE, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA,
MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PHAR, PHARMASEARCH, PROMT, RTECS*,
SPECINFO, TOXCENTER, ULIDAT, USAN, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



Sirolimus

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

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18594 REFERENCES IN FILE CAPLUS (1967 TO DATE)
186 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> s cyclosporin
L4 1126 CYCLOSPORIN

=> s cyclosporin/cn
L5 2 CYCLOSPORIN/CN

=> d

L5 ANSWER 1 OF 2 REGISTRY COPYRIGHT 2002 ACS
RN 79217-60-0 REGISTRY
CN **Cyclosporin (9CI)** (CA INDEX NAME)
MF Unspecified
CI COM, MAN
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO,
CA, CABA, CAPLUS, CBNB, CEN, CHEMLIST, CIN, DIOGENES, EMBASE, MSDS-OHS,
NIOSHTIC, PROMT, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
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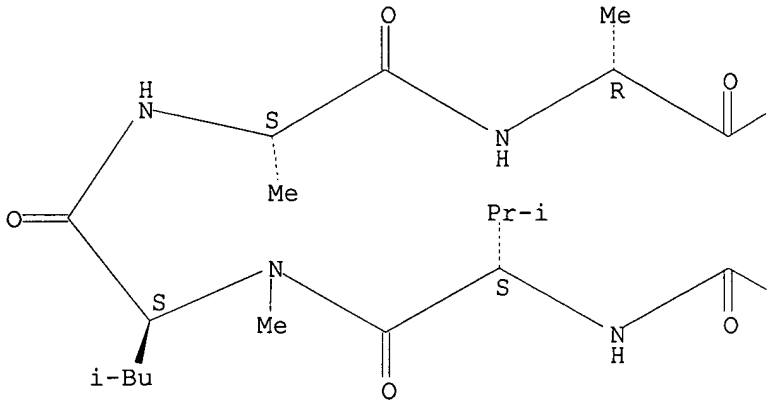
60 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
676 REFERENCES IN FILE CAPLUS (1967 TO DATE)

=> d 2

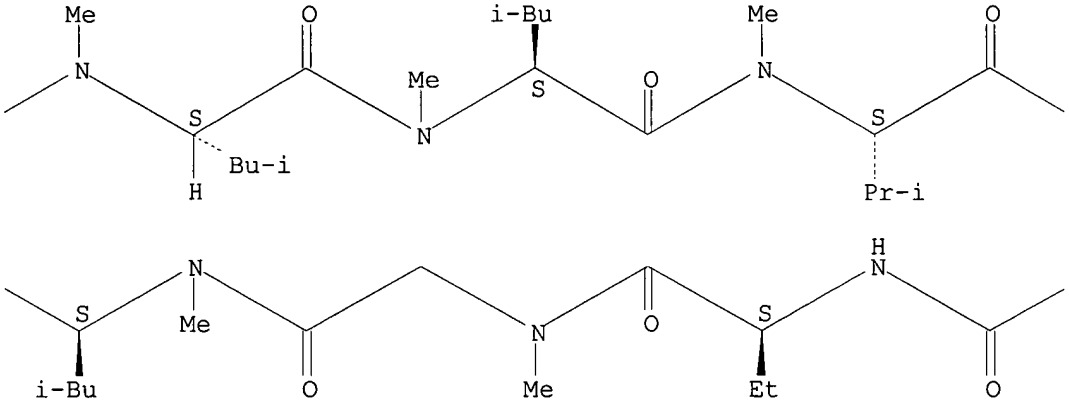
L5 ANSWER 2 OF 2 REGISTRY COPYRIGHT 2002 ACS
RN 59865-13-3 REGISTRY
CN Cyclosporin A (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 1,4,7,10,13,16,19,22,25,28,31-Undecaazacyclotritriacontane, cyclic peptide
deriv.
OTHER NAMES:
CN 7: PN: WO0002548 PAGE: 30 claimed protein
CN Antibiotic S 7481F1
CN Ciclosporin
CN Cipol N
CN Consupren
CN **Cyclosporin**
CN Cyclosporine
CN Cyclosporine A
CN Cyclo[L-alanyl-D-alanyl-N-methyl-L-leucyl-N-methyl-L-leucyl-N-methyl-L-valyl-(3R,4R,6E)-6,7-didehydro-3-hydroxy-N,4-dimethyl-L-2-aminooctanoyl-L-2-aminobutanoyl-N-methylglycyl-N-methyl-L-leucyl-L-valyl-N-methyl-L-leucyl]
CN Neoplanta
CN Neoral
CN OL 27-400
CN Ramihyphin A
CN S-Neoral
CN Sandimmun
CN Sandimmun Neoral
CN Sandimmune
CN Sang-35
CN SDZ-OXL 400
FS PROTEIN SEQUENCE; STEREOSEARCH
DR 56645-58-0, 55126-45-9, 104250-72-8, 223528-56-1
MF C62 H111 N11 O12
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS,
BIOTECHNO, CA, CABA, CANCERLIT, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DIOGENES, DRUGNL, DRUGPAT, DRUGU, DRUGUPDATES, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PHAR, PHARMASEARCH, PROMT, RTECS*, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: WHO

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



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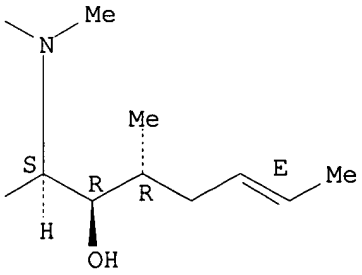


11214 REFERENCES IN FILE CA (1967 TO DATE)
292 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

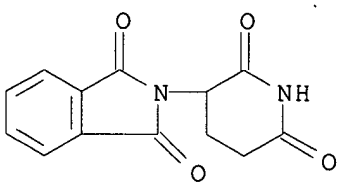
11233 REFERENCES IN FILE CAPLUS (1967 TO DATE)

=> s triamcinalone acetonide/cn
L6 0 TRIAMCINALONE ACETONIDE/CN

=> s thalidomide/cn
L7 1 THALIDOMIDE/CN

=> d

L7 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2002 ACS
RN 50-35-1 REGISTRY
CN 1H-Isoindole-1,3(2H)-dione, 2-(2,6-dioxo-3-piperidinyl)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Phthalimide, N-(2,6-dioxo-3-piperidyl)- (6CI, 7CI, 8CI)
OTHER NAMES:
CN (.+-.)-Thalidomide
CN .alpha.-(N-Phthalimido)glutarimide
CN .alpha.-N-Phthalylglutaramide
CN .alpha.-Phthalimidoglutarimide
CN 1,3-Dioxo-2-(2,6-dioxopiperidin-3-yl)isoindoline
CN 3-Phthalimidoglutarimide
CN Celgene
CN Contergan
CN Distaval
CN K 17
CN Kevadon
CN N-(2,6-Dioxo-3-piperidyl)phthalimide
CN N-Phthaloylglutamimide
CN Quetimid
CN Sedoval
CN Softenil
CN Softenon
CN Talimol
CN **Thalidomide**
CN Thalomid
FS 3D CONCORD
DR 14088-68-7, 731-40-8
MF C13 H10 N2 O4
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DIOGENES, DRUGNL, DRUGU, DRUGUPDATES, EMBASE, HODOC*, HSDB*, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PHAR, PHARMASEARCH, PIRA, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USAN, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)



514/339

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**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

             821 REFERENCES IN FILE CA (1967 TO DATE)
              49 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
             828 REFERENCES IN FILE CAPLUS (1967 TO DATE)
              15 REFERENCES IN FILE CAOLD (PRIOR TO 1967)


=> file embasebiosis medline caplus uspatfull
'EMBASEBIOSIS' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available.  If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):embase biosis
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                41.34      41.55

FILE 'EMBASE' ENTERED AT 17:13:21 ON 11 JUN 2002
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FILE 'MEDLINE' ENTERED AT 17:13:21 ON 11 JUN 2002

FILE 'CAPLUS' ENTERED AT 17:13:21 ON 11 JUN 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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FILE 'USPATFULL' ENTERED AT 17:13:21 ON 11 JUN 2002
CA INDEXING COPYRIGHT (C) 2002 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOSIS' ENTERED AT 17:13:21 ON 11 JUN 2002
COPYRIGHT (C) 2002 BIOLOGICAL ABSTRACTS INC.(R)

=> s fluocinonide or decadron or cyclosporin or 356-12-7/rn or 61-76-7/rn or
50-02-2/rn or 79217-60-0/rn
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
L8      125490 FLUOCINONIDE OR DECADRON OR CYCLOSPORIN OR 356-12-7/RN OR
61-76-
               7/RN OR 50-02-2/RN OR 79217-60-0/RN

=> s aphthous or aphthae or oral ulcer or oral lesion
L9       10325 APHTHOUS OR APHTHAE OR ORAL ULCER OR ORAL LESION

=> s l8 and l9
L10        205 L8 AND L9

=> s thalidomide or PTX or 50-35-1/rn or 6493-05-6/rn
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
L11      22276 THALIDOMIDE OR PTX OR 50-35-1/RN OR 6493-05-6/RN

=> s l10 and l11
L12         54 L10 AND L11
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L13   34 L12 AND PY<2000

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PROCESSING COMPLETED FOR L13
L14   31 DUP REM L13 (3 DUPLICATES REMOVED)

=> d l14 1-31 ab bib kwic

L14 ANSWER 1 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB   The tenacious effort to develop new, specific agents to treat HIV
     infection is currently accompanied by a reconsideration of existing drugs
     on the basis of their known or putative effects on the retroviral life
     cycle and/or the tuning of immune mechanisms. Three specific 'older'
     compounds that interfere with HIV infection by both a direct antiviral
     activity, and a modulation of T-cell activation and proliferation have
     received the most attention. Hydroxurea, a classical chemotherapeutic
     agent, inhibits retroviral reverse transcription by targeting a cellular
     enzyme responsible for the synthesis of deoxynucleoside triphosphates. It
     may also have a role in reducing viral load while maintaining low numbers
     of potential target T cells. Beneficial effects of hydroxyurea in
     combination with didanosine and/or stavudine on viral load have been
shown
     in a number of clinical trials. **Cyclosporin**, a known
     immunosuppresant, blocks the activation of T cells, hence reducing the
     permissivity to HIV, and also prevents proper HIV virion maturation.
     However, clinical studies have produced conflicting results in
     HIV-infected patients with regard to immunological and disease effects
and
     toxicity. **Thalidomide** may have antiretroviral effects as a
     result of its primarily inhibitory effects on the production of tumour
     necrosis factor .alpha. (TNF.alpha.). TNF.alpha. induces expression of
HIV
     from chronically infected cell lines by stimulating a cellular
     transcription factor, and blocking of TNF.alpha.-stimulated HIV
     replication by **thalidomide** has been shown in vitro and ex vivo.
     However, the effects on TNF.alpha. production in vivo have been
     inconsistent. **Thalidomide** has shown potential in treating some
     AIDS-related conditions [cachexia (weight loss and muscle wasting), and
     aphtous oral, oesophageal or genital ulcers]. However, because of its
     numerous and major adverse effects, **thalidomide** should always be
     used cautiously. In summary, some older drugs have potential as anti-HIV
     agents and offer the advantage of extensive clinical experience in other
     therapeutic areas. They should be considered as potential partners for
the
     products emerging from more recent research and development.
AN   2000037704 EMBASE
TI   New uses for old drugs in HIV infection. The role of hydroxyurea,
     cyclosporin and **thalidomide**.
AU   Ravot E.; Lisziewicz J.; Lori F.
CS   Dr. F. Lori, Res. Inst. Genetic and Human Therapy, Policlinico San
Matteo,
     Padiglione Forlanini, P. le Golgi 2, 27100 Pavia, Italy.
     RIGHT@gunet.georgetown.edu
SO   Drugs, (1999) 58/6 (953-963).
     Refs: 70
     ISSN: 0012-6667 CODEN: DRUGAY
CY   New Zealand
DT   Journal; General Review

FS 030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
TI New uses for old drugs in HIV infection. The role of hydroxyurea, **cyclosporin** and **thalidomide**.
SO Drugs, (1999) 58/6 (953-963).
Refs: 70
ISSN: 0012-6667 CODEN: DRUGAY
AB . . . of hydroxyurea in combination with didanosine and/or stavudine on viral load have been shown in a number of clinical trials. **Cyclosporin**, a known immunosuppresant, blocks the activation of T cells, hence reducing the permissivity to HIV, and also prevents proper HIV. . . maturation. However, clinical studies have produced conflicting results in HIV-infected patients with regard to immunological and disease effects and toxicity. **Thalidomide** may have antiretroviral effects as a result of its primarily inhibitory effects on the production of tumour necrosis factor .alpha.. . . of HIV from chronically infected cell lines by stimulating a cellular transcription factor, and blocking of TNF.alpha.-stimulated HIV replication by **thalidomide** has been shown in vitro and ex vivo. However, the effects on TNF.alpha. production in vivo have been inconsistent. **Thalidomide** has shown potential in treating some AIDS-related conditions [cachexia (weight loss and muscle wasting), and aphtous oral, oesophageal or genital ulcers]. However, because of its numerous and major adverse effects, **thalidomide** should always be used cautiously. In summary, some older drugs have potential as anti-HIV agents and offer the advantage of. . .
CT Medical Descriptors:
*Human immunodeficiency virus infection: DT, drug therapy
life cycle
Retrovirus
immunity
antiviral activity
T lymphocyte activation
lymphocyte proliferation
reverse transcription
virus load
T lymphocyte
cell count
virion
maturation
cytokine production
virus replication
cachexia: CO, complication
aphthous ulcer: CO, complication
mouth ulcer: CO, complication
genital ulcer: CO, complication
esophagus ulcer: CO, complication
side effect: EP, epidemiology
side effect: ET, etiology
side effect: SI, side effect
review
*hydroxyurea: CB, drug combination
*hydroxyurea: DT, drug therapy
*hydroxyurea: PD, pharmacology
***cyclosporin: DT, drug therapy**

***cyclosporin: PD, pharmacology**
***thalidomide: AE, adverse drug reaction**
***thalidomide: DT, drug therapy**
***thalidomide: PD, pharmacology**
nucleoside triphosphate: EC, endogenous compound
didanosine: CB, drug combination
stavudine: CB, drug combination
tumor necrosis factor alpha: EC, endogenous compound
transcription factor: EC,. . .
RN (hydroxyurea) 127-07-1; (**cyclosporin**) 79217-60-0; (**thalidomide**) 50-35-1; (didanosine) 69655-05-6; (stavudine) 3056-17-5

L14 ANSWER 2 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 1
AB Behcet's syndrome (BS), originally described as a triad of oral **aphthae**, genital ulcerations and uveitis, is a systemic vasculitis that is prevalent in the Middle east, Far East and in the Mediterranean basin. It is characterized by a heightened state of inflammation although the main drive that initiates and sustains this is not yet elucidated. Suppression of this inflammatory state constitutes the major goal of treatment and therapy is tailored according to the specific manifestations observed. We now have considerable more insight on drug management of BS compared to 20 years ago. Particularly, within the recent past we have learned to use more rationally the agents that were already available to us. This is especially true for azathioprine, **cyclosporin** A, **thalidomide** and colchicine. Promising data are also being collected with alpha-interferon. With these agents, significant progress has been achieved in the management of uveitis and mucocutaneous symptoms but treatment issues related to thrombotic problems, major vessel involvement and neurological disease have not yet been resolved.
AN 2000014648 EMBASE
TI The management of Behcet's syndrome.
AU Fresko I.; Yurdakul S.; Hamuryudan V.; Ozyazgan Y.; Mat C.; Tanverdi M.M.; Yazici H.
CS H. Yazici, Department of Internal Medicine, Cerrahpasa Medical Faculty, University of Istanbul, Aksaray-Istanbul 34303, Turkey. hyazici@ibm.net
SO Annales de Medecine Interne, (1999) 150/7 (576-581).
Refs: 47
ISSN: 0003-410X CODEN: AMDIBO
CY France
DT Journal; General Review
FS 006 Internal Medicine
037 Drug Literature Index
LA English
SL English; French
SO Annales de Medecine Interne, (1999) 150/7 (576-581).
Refs: 47
ISSN: 0003-410X CODEN: AMDIBO
AB Behcet's syndrome (BS), originally described as a triad of oral **aphthae**, genital ulcerations and uveitis, is a systemic vasculitis that is prevalent in the Middle east, Far East and in the. . . have learned to use more rationally the agents that were already available to us. This is especially true for azathioprine, **cyclosporin** A, **thalidomide** and colchicine. Promising data are also being collected with alpha-interferon. With these agents, significant progress has been achieved in the. . .
CT Medical Descriptors:
*Behcet disease

clinical feature
aphthous stomatitis
genital ulcer
uveitis
treatment planning
immunosuppressive treatment
review
*azathioprine
***cyclosporin A**
***thalidomide**
*colchicine
RN (azathioprine) 446-86-6; (**cyclosporin** A) 59865-13-3, 63798-73-2; (**thalidomide**) 50-35-1; (colchicine) 64-86-8

L14 ANSWER 3 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 1999369478 EMBASE
TI Behcet's disease in the Middle East.
AU Saylan T.; Mat C.; Fresko I.; Melikoglu M.
CS Prof. C. Mat, Dermatoloji Anabilim Dali Cerrahpasa, Cerrahpasa Tip Fakultesi, Istanbul Universitesi, Istanbul 34303, Turkey
SO Clinics in Dermatology, (1999) 17/2 (209-223).
Refs: 123
ISSN: 0738-081X CODEN: CLDEEU
PUI S 0738-081X(99)00013-9
CY United States
DT Journal; General Review
FS 012 Ophthalmology
013 Dermatology and Venereology
017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
LA English
SO Clinics in Dermatology, (1999) 17/2 (209-223).
Refs: 123
ISSN: 0738-081X CODEN: CLDEEU
CT Medical Descriptors:
*Behcet disease: ET, etiology
*Behcet disease: EP, epidemiology
*Behcet disease: DT, drug therapy
*Behcet disease: DI, diagnosis
prevalence
pathogenesis
clinical feature
aphthous stomatitis: CO, complication
genital ulcer: CO, complication
differential diagnosis
histopathology
human
oral drug administration
review
priority journal
azathioprine: DT, drug therapy
cyclosporin: DT, drug therapy
thalidomide: DT, drug therapy
corticosteroid: DT, drug therapy
nonsteroid antiinflammatory agent: DT, drug therapy
cyclophosphamide: DT, drug therapy
salazosulfapyridine: DT, drug therapy
alpha interferon: DT, drug. . .
RN (azathioprine) 446-86-6; (**cyclosporin**) 79217-60-0; (

thalidomide) 50-35-1; (cyclophosphamide) 50-18-0;
(salazosulfapyridine) 599-79-1

L14 ANSWER 4 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 1999132215 EMBASE
TI [Extraintestinal mucocutaneous manifestations of chronic inflammatory bowel diseases].
MANIFESTATIONS CUTANEO-MUQUEUSES EXTRA-INTESTINALES DES MALADIES INFLAMMATOIRES CHRONIQUES DE L'INTESTIN.
AU Bonnet J.; Roux M.-E.; Rybojad M.; Lemann M.
CS M. Lemann, Service de Dermatologie, Hopital Saint-Louis, 1, avenue Claude-Velle-Faux, 75010 Paris, France
SO Hepato-Gastro, (1999) 6/2 (113-121).
Refs: 38
ISSN: 1253-7020 CODEN: HEGAF6
CY France
DT Journal; (Short Survey)
FS 013 Dermatology and Venereology
037 Drug Literature Index
038 Adverse Reactions Titles
048 Gastroenterology
LA French
SO Hepato-Gastro, (1999) 6/2 (113-121).
Refs: 38
ISSN: 1253-7020 CODEN: HEGAF6
CT Medical Descriptors:
*enteritis
clinical feature
prevalence
pyoderma gangrenosum: DT, drug therapy
pyoderma gangrenosum: ET, etiology
skin biopsy
aphthous stomatitis: DT, drug therapy
aphthous stomatitis: ET, etiology
erythema nodosum: ET, etiology
acute febrile neutrophilic dermatosis: ET, etiology
epidermolysis bullosa acquisita: ET, etiology
skin disease: SI, side effect
human
short survey
*acetylsalicylic acid: DT, drug therapy
*lidocaine: DT, drug therapy
*betamethasone valerate: DT, drug therapy
*tetracycline derivative: DT, drug therapy
***thalidomide: DT, drug therapy**
*colchicine: DT, drug therapy
indometacin: DT, drug therapy
prednisone: DT, drug therapy
methylprednisolone: DT, drug therapy
dapsone: DT, drug therapy
salazosulfapyridine: AE, adverse drug reaction
salazosulfapyridine: DT, drug therapy
mesalazine: AE, adverse drug reaction
azathioprine: AE, adverse drug reaction
mercaptopurine: AE, adverse drug reaction
cyclosporin: AE, adverse drug reaction
methotrexate: AE, adverse drug reaction
metronidazole: AE, adverse drug reaction
ciprofloxacin: AE, adverse drug reaction
RN (acetylsalicylic acid) 493-53-8, 50-78-2, 53663-74-4, 53664-49-6,

63781-77-1; (lidocaine) 137-58-6, 24847-67-4, 56934-02-2, 73-78-9; (betamethasone valerate) 2152-44-5, 57654-97-4; (**thalidomide**) 50-35-1; (colchicine) 64-86-8; (indometacin) 53-86-1, 74252-25-8, 7681-54-1; (prednisone) 53-03-2; (methylprednisolone) 6923-42-8, 83-43-2; (dapsone) 80-08-0; (salazosulfapyridine) 599-79-1; (mesalazine) 89-57-6; (azathioprine) 446-86-6; (mercaptopurine) 31441-78-8, 50-44-2, 6112-76-1; (**cyclosporin**) 79217-60-0; (methotrexate) 15475-56-6, 59-05-2, 7413-34-5; (metronidazole) 39322-38-8, 443-48-1; (ciprofloxacin) 85721-33-1

L14 ANSWER 5 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AB Behcet's disease is a multisystem disorder with the histological picture of a leukocytoclastic vasculitis. It's main features are orogenital ulcerations (**aphthae**), skin changes and an oligoarthritis, as well as anterior and posterior uveitis (mainly a retinal vasculitis) and arterial/venous thrombosis or aneurysm. Due to the manifold symptoms and the unsatisfactory therapeutic results especially concerning the ocular manifestations, the disease is a challenge for the rheumatologist. Above all, a good cooperation with ophthalmologists, dermatologists and vascular surgeons is necessary. This review article describes the manifestations, diagnostic criteria and therapeutic options in Behcet's disease.

AN 1999218224 EMBASE

TI [Current aspects of diagnostik criteria and therapeutic options in Behcet's disease].
AKTUELLE ASPEKTE DER DIAGNOSTIK UND THERAPIE DES MORBUS BEHCET.

AU Kotter I.; Stubiger N.
CS Dr. I. Kotter, Medizinische Universitatsklinik, Abteilung Innere Medizin II, Ottfried-Muller-Strasse 10, D-72 076 Tubingen, Germany

SO Aktuelle Rheumatologie, (1999) 24/2 (51-57).
Refs: 48
ISSN: 0341-051X CODEN: AKRHDB
CY Germany
DT Journal; Article
FS 011 Otorhinolaryngology
012 Ophthalmology
013 Dermatology and Venereology
031 Arthritis and Rheumatism
037 Drug Literature Index
LA German
SL English; German

SO Aktuelle Rheumatologie, (1999) 24/2 (51-57).
Refs: 48
ISSN: 0341-051X CODEN: AKRHDB
AB Behcet's disease is a multisystem disorder with the histological picture of a leukocytoclastic vasculitis. It's main features are orogenital ulcerations (**aphthae**), skin changes and an oligoarthritis, as well as anterior and posterior uveitis (mainly a retinal vasculitis) and arterial/venous thrombosis or. . .

CT Medical Descriptors:
*Behcet . . . drug therapy
arthritis: CO, complication
arthritis: DT, drug therapy
artery thrombosis: CO, complication
artery thrombosis: DT, drug therapy
vein thrombosis: CO, complication
vein thrombosis: DT, drug therapy
aphthous ulcer: CO, complication
aphthous ulcer: DT, drug therapy
immunosuppressive treatment

human
oral drug administration
article
demecolcine: DT, drug therapy
thalidomide: DT, drug therapy
prednisolone: DT, drug therapy
heparin: DT, drug therapy
cyclosporin a: DT, drug therapy
cyclophosphamide: DT, drug therapy
chlorambucil: DT, drug therapy
alpha2a interferon: DT, drug therapy
tsukubaenolide: DT, drug therapy
RN (demecolcine) 477-30-5; (**thalidomide**) 50-35-1; (prednisolone) 50-24-8; (heparin) 37187-54-5, 8057-48-5, 8065-01-8, 9005-48-5; (**cyclosporin** a) 59865-13-3, 63798-73-2; (cyclophosphamide) 50-18-0; (chlorambucil) 305-03-3; (alpha2a interferon) 76543-88-9; (tsukubaenolide) 104987-11-3

L14 ANSWER 6 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB Behcet's disease is a complex multisystem disease diagnosed by means of clinical criteria. Clinical features include oral and genital **aphthae**, pustular vasculitic cutaneous lesions, and ocular, gastrointestinal, and vascular manifestations. We believe that complex aphthosis, characterized by oral or oral and genital ulcers, may be a forme fruste of Behcet's disease. Although the pathogenesis of both Behcet's disease and complex aphthosis remain unknown, immune factors, infectious agents, and effector mechanisms are implicated. Treatment is based on the severity of systemic involvement and includes topical therapies as well as colchicine, dapsone, **thalidomide**, and immunosuppressive agents.
AN 1999039220 EMBASE
TI Behcet's disease and complex aphthosis.
AU Ghate J.V.; Jorizzo J.L.
CS Dr. J.L. Jorizzo, Department of Dermatology, Wake Forest Univ. School of Medicine, Medical Center Blvd, Winston-Salem, NC 27157, United States
SO Journal of the American Academy of Dermatology, (1999) 40/1 (1-18). Refs: 252
ISSN: 0190-9622 CODEN: JAADDB
CY United States
DT Journal; General Review
FS 011 Otorhinolaryngology
013 Dermatology and Venereology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
SO Journal of the American Academy of Dermatology, (1999) 40/1 (1-18). Refs: 252
ISSN: 0190-9622 CODEN: JAADDB
AB Behcet's disease is a complex multisystem disease diagnosed by means of clinical criteria. Clinical features include oral and genital **aphthae**, pustular vasculitic cutaneous lesions, and ocular, gastrointestinal, and vascular manifestations. We believe that complex aphthosis, characterized by oral or oral. . . are implicated. Treatment is based on the severity of systemic involvement and includes topical therapies as well as colchicine, dapsone, **thalidomide**, and immunosuppressive agents.

CT Medical Descriptors:
*Behcet disease: DI, diagnosis
*Behcet disease: DT, drug therapy
*Behcet disease: EP, epidemiology
*Behcet disease: ET, etiology
***aphthous ulcer: DI, diagnosis**
***aphthous ulcer: DT, drug therapy**
***aphthous ulcer: EP, epidemiology**
***aphthous ulcer: ET, etiology**
aphthous stomatitis: DI, diagnosis
aphthous stomatitis: DT, drug therapy
aphthous stomatitis: EP, epidemiology
aphthous stomatitis: ET, etiology
genital ulcer: DI, diagnosis
genital ulcer: DT, drug therapy
genital ulcer: EP, epidemiology
genital ulcer: ET, etiology
uveitis: DI, diagnosis
uveitis: DT, drug. . . journal
*immunosuppressive agent: AE, adverse drug reaction
*immunosuppressive agent: DT, drug therapy
*colchicine: AE, adverse drug reaction
*colchicine: DT, drug therapy
*dapsone: DT, drug therapy
***thalidomide: AE, adverse drug reaction**
***thalidomide: CT, clinical trial**
***thalidomide: DT, drug therapy**
corticosteroid: AE, adverse drug reaction
corticosteroid: CT, clinical trial
corticosteroid: DT, drug therapy
methotrexate: DT, drug therapy
prednisone: CT, clinical trial
prednisone: DT,. . . AE, adverse drug reaction
alpha2a interferon: DT, drug therapy
azathioprine: AE, adverse drug reaction
azathioprine: DT, drug therapy
cyclophosphamide: DT, drug therapy
chlorambucil: DT, drug therapy
cyclosporin: AE, adverse drug reaction
cyclosporin: DT, drug therapy
clobetasol: DT, drug therapy
tetracycline: DT, drug therapy
RN (colchicine) 64-86-8; (dapsone) 80-08-0; (**thalidomide**) 50-35-1; (methotrexate) 15475-56-6, 59-05-2, 7413-34-5; (prednisone) 53-03-2; (triamcinolone) 124-94-7; (alpha2a interferon) 76543-88-9; (azathioprine) 446-86-6; (cyclophosphamide) 50-18-0; (chlorambucil) 305-03-3; (**cyclosporin**) 79217-60-0; (clobetasol) 25122-41-2; (tetracycline) 23843-90-5, 60-54-8, 64-75-5

L14 ANSWER 7 OF 31 USPATFULL
AB Methods of treatment for inflammatory and autoimmune dermatoses which comprises topical and/or systemic administration of a therapeutically-effective amount of **thalidomide** alone or in combination with other dermatological agents.
AN 97:68480 USPATFULL
TI Treatment of inflammatory and/or autoimmune dermatoses with **thalidomide** alone or in combination with other agents
IN Andrulis, Jr., Peter J., Bethesda, MD, United States
Drulak, Murray W., Gaithersburg, MD, United States
PA Andrulis Pharmaceuticals, Beltsville, MD, United States (U.S.

corporation)
PI US 5654312 19970805 <--
AI US 1995-475426 19950607 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Nutter, Nathan M.
LREP Angres, Isaac
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 925
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Treatment of inflammatory and/or autoimmune dermatoses with **thalidomide** alone or in combination with other agents
PI US 5654312 19970805 <--
AB Methods of treatment for inflammatory and autoimmune dermatoses which comprises topical and/or systemic administration of a therapeutically-effective amount of **thalidomide** alone or in combination with other dermatological agents.
SUMM The present invention relates to novel methods for treating inflammatory and/or autoimmune dermatoses with **thalidomide** alone or in combination with other agents. The present invention also relates to methods of treating dermatoses with inhibitors of. . .
SUMM . . . be triggered by a number of external events ranging from exposure to UV light from the sun to an allergen. **Thalidomide** has been demonstrated to have an inhibitory effect on the pro-inflammatory cytokines. It has been shown to inhibit TNF-alpha production. . . stimulated monocytes (Sampaio et al., J. Exp. Med., 173:699-703, 1991). Moreira et al. (J. Exp. Med., 177:1675-80, 1993) reported that **thalidomide** acts by enhancing TNF-alpha m-RNA degradation. Shannon et al. (Amer. Society for Microbiology Ann. Mtg., Abs. U-53, 1990) indicated **thalidomide** inhibited IL-1 beta production in vitro. Such an inhibitory effect on IL-1 beta may be direct or indirect through TNF-alpha. . .
SUMM . . . surface of endothelial cells facilitates the binding of inflammatory cells that is a precondition to transendothelial migration occurring during inflammation. **Thalidomide** also has an anti-angiogenic effect since TNF-alpha stimulates endothelial cell motility in vitro (Leibovich, Nature, 329:630-32, 1987; Rosen et al.,. . . et al., Proc. Natl. Acad. Sci. (USA), 84:5277-5291, 1987). D'Amato et al. (Proc. Natl. Acad. Sci. (USA), 91:4082-5,1994) showed that **thalidomide** was an effective inhibitor of angiogenesis induced by bFGF.
SUMM In 1965 Sheskin (Lepr. Rev., 36:183-7) administered **thalidomide** to leprosy patients suffering from the complication erythema nodosum leprosum (ENL), to sedate them. ENL is characterized by recurrent erythematosus nodules on the skin, weight loss, mania, neuritis, fever, malaise, and sometimes epididyo-orchitis. Within 12 hours of **thalidomide** administration nodular eruptions began to heal and within two days fever declined and the ENL lesions had completely resolved. In. . . double blind clinical trial conducted in four countries and coordinated by the World Health Organization, which tested the efficacy of **thalidomide** versus aspirin for treatment of ENL. The conclusions reached supported Sheskin's original observations about the effectiveness of **thalidomide** for treatment of ENL. Wemambu et al. (Lancet, 2:933-5, 1969) observed necrotizing vasculitis of veins and arteries in patients with. . . Appl. Immun., 57:317-332

(1978) showed in a study of neutrophil activation in ENL patients just before and during treatment with **thalidomide** that tissue damage was not due solely to neutrophil activation as occurs in immune complex diseases, but rather neutrophils appeared to be activated by an undefined lymphokine. This group went on to state that the therapeutic effect of **thalidomide** was not due to inhibition of neutrophil activation. Sarno et al. (Clin. Exp. Immunol., 84:103-8, 1991) showed that TNF-alpha levels were elevated in ENL patients and that TNF-alpha had a major role in the pathogenesis of this disease. **Thalidomide** was shown to inhibit TNF-alpha production in these ENL patients. Sampaio et al. (J. Inf. Dis., 168:408-14, 1993) confirmed Sarno's. . .

SUMM The fortuitous finding that **thalidomide** was effective in treating ENL stimulated other investigators to look at the efficacy of **thalidomide** for treating other dermatoses with a possible inflammatory and/or autoimmune pathogenesis.

SUMM . . . areas of the body. Its etiology is unknown. Londono (Int. J. Dermatol., 12:326-8, 1973) was the first to report using **thalidomide** as a treatment for actinic prurigo. He administered 300 mg of **thalidomide** per day to 34 patients until clinical improvement was noted and then reduced the dosage progressively. There was notable improvement. . . an immunological etiology. Lovell et al.

(Brit. J. Dermatol, 108:467-71, 1983) treated 14 actinic prurigo patients with 50-100 mg of **thalidomide** per day for children and 100-200 mg of **thalidomide** per day for adults, for variable periods of time. Eleven patients had long term clinical improvement and three were free of symptoms even after **thalidomide** was discontinued. No side effects were noted.

SUMM . . . on the basis of clinical criteria. Mattos (Bol. Div. Nac. Lepra., 32:71) in 1973 was the first investigator to use **thalidomide** to treat prurigo nodularis. One of the two patients treated received 200 mg per day of **thalidomide** and the other patient, a woman, received 300 mg daily. Both patients had excellent clinical responses to the therapy after several weeks. Sheskin (Hautarzt, 26:215, 1975) reported treating three prurigo nodularis patients with **thalidomide**. These patients suffered from the disease for eight to twenty-four years, but responded clinically within a few weeks of initiation of **thalidomide** therapy. Other studies (Van den Broek, Arch. Dermatol, 116:571, 1980; Nikolowski, Hautarzt, 31:565, 1980; Winkelmann et al., Acta. Dermato-Venereologica, 64:412-7,. . . the intensive itch that accompanies this condition subsiding within 2-3 weeks of the start of 200 mg per day of **thalidomide**. However, in these studies it was noted that it takes at least six months of **thalidomide** therapy before strongly lichenified lesions completely heal.

SUMM . . . certain drugs. Barba-Rubio and Gonzalez, Derm. Rev. Mex., 19:131 (1975) treated 20 discold lupus erythematosus patients with 300 mg of **thalidomide** per day. Within two weeks 19 of these patients responded clinically and the medication was then reduced to a maintenance. . . al., Giorn. Ital. Derre. Vener, 115:471, 1980; Samsoen et al., Ann. Dermatol Venereol (Paris), 107:515-23, 1980) confirmed the effectiveness of **thalidomide** therapy in treating discold lupus erythematosus patients refractory to other treatments such

as steroids. In most instances a clinical effect was detected within 14 days of initiation of 100-200 mg per day of **thalidomide**, however, a total and definite recovery was seen in only 15-20% of patients. In most patients a 25-50 mg per day maintenance dose of **thalidomide** was required to sustain a clinical improvement.

SUMM **Thalidomide** has also been used successfully to treat Behcet's syndrome, a rare and severe illness of unknown etiology often afflicting young. . . and genitalia, uveitis, and retinal vasculitis. There also may be atrophy of the gastrointestinal tract and pulmonary or myocardial fibrosis. **Thalidomide** therapy was an important breakthrough, because prior to this there was no specific treatment for Behcet's syndrome. Steroids proved to. . . prescribed (Mamo et al., Arch. Ophthamol, 71:4-14, 1964). Saylan and Saltik (Arch. Dermatol, 118:536, 1982) were the first to use **thalidomide** to treat 22 patients with Behcet's syndrome who had deep and persistent oral **aphthae** . Patients were initially administered 400 mg per day of **thalidomide** for five days followed by 200 mg per day for 15 to 60 days. This regimen resulted in rapid and complete healing of **aphthae**. Torras et al. (Arch. Dermatol, 118:875, 1982) found that there was complete healing of giant **aphthae** in eight of nine Behcet's patients treated with 100 mg per day of **thalidomide** for 10 days. Jorizzo et al. (Arch. Int. Med., 146:878-81, 1986) reported similar success with **thalidomide** in five patients with Behcet's syndrome. In 1993 Denman et al., Rev. Med. Int., 14:(suppl 1) 495, treated 39 patients with Behcet's syndrome with 50 mg of **thalidomide** three nights per week for a mean time of 35.9 months and a maximum treatment time of up to 65 months. Concomitant treatment in this patient group included 10 patients on prednisone, 3 on azathioprine and 1 patient on **cyclosporin**. Mucosal lesions healed in all patients. Moulin et al. (Ann. Dermatol Venereol, 110:611, 1983) used 100 mg per day of **thalidomide** to treat six patients with a Jessner-Kanof lymphocytic infiltration of the skin. This disease is characterized by numerous lesions on. . . i:251 (1977) treated a patient with a relapsing non-suppurative panniculitis termed Weber Christian Disease, with 300 mg per day of **thalidomide** for three weeks which was reduced to 200 mg per day and then to 100 mg per day after 10. . . lesions steadily regressed during therapy and it was reported that a disease free state was maintained for 13 months after **thalidomide** was stopped. **Thalidomide** has also been used to treat recurrent erythema multiforme, a flu like syndrome in which blisters appear on mucous membranes. . . Bahmer et al., Acta. Derm. Venereal, 62:449 (1982) treated a patient who had recurrent erythema multiforme with 200 mg of **thalidomide** per day. Within a few days the mucosal membrane and skin lesions healed and the daily dosage of **thalidomide** was lowered. The patient has been maintained in a disease free state by administration of 100 mg of **thalidomide** per day.

SUMM As indicated oral administration of **thalidomide** has been successfully used to treat a limited number of dermatoses that may have an autoimmune and/or inflammatory component associated with them. Topical application of **thalidomide** is a useful therapeutic approach for disease states with an autoimmune and/or inflammatory basis. Furthermore, **thalidomide** may be used alone to treat dermatoses with an autoimmune and/or inflammatory basis or in unique combinations with other cytokine/growth. . . anti-inflammatory and/or anti auto-immune agents and/or other physical and/or chemical dermatological treatments. An example of such combination therapy could involve **thalidomide** given with pentoxifylline and a

glucocorticoid such as dexamethasone. The activity of each of these agents would be expected to. . . a different point in this synthesis.

Pentoxifylline inhibits TNF-alpha gene transcription (Doherty et al., Surgery (St. Louis), 110:192, 1991), while **thalidomide** enhances TNF-alpha m-RNA degradation (Moreira et al., 1993) and glucocorticoids such as dexamethasone inhibit TNF-alpha m-RNA translation (Han et al.,. . .

SUMM **Thalidomide** has been administered orally, however, it may be used topically to treat dermatoses with an autoimmune and/or inflammatory component associated. . .

SUMM **Thalidomide** was first synthesized and marketed in the 1950's as a sedative. The toxicity of the compound was so low that a dose killing 50% of animals (LD.sub.50) could not be established. **Thalidomide** was therefore thought to be a safer alternative to barbiturates. In 1961 **thalidomide** administered to pregnant women resulted in an epidemic of congenital malformation. The incidence of malformed babies paralleled the sales of **thalidomide** and quickly dropped off when **thalidomide** was removed from the market.

SUMM Oral administration of **thalidomide** in the range of 100-200 mg in adult humans results in a peak blood level of 0.9-1.5 mg/liter after 4-6 hours. Hydrolytic cleavage of **thalidomide** occurs in vitro, the rate of which increases as the pH increases. However, hydrolytic cleavage of **thalidomide** in serum is much slower than in vitro at pH 7.4. This may be due to **thalidomide** being highly bound to plasma proteins. Studies in animals demonstrated high **thalidomide** concentrations in the gastrointestinal tract, liver and kidneys with lower concentrations in muscle, brain and adipose tissue. In pregnant animals, **thalidomide** can pass across the placenta.

SUMM Although a complete study of **thalidomide** metabolism in humans has not been performed, in animals the main pathway for **thalidomide** breakdown appears to be nonenzymatic hydrolytic cleavage. Even though immunomodulatory effects of **thalidomide** have not been clearly defined at the molecular level, **thalidomide** has been used to treat the following immunologically-based diseases: acute **aphthous** ulcers (Jenkins et al., Lancet, 2:1424-6, 1984; Grinspan, J. Amer. Acad. Dermatol, 12:85-90, 1985; Revuz et al., Arch. Dermatol, 126:923-7,. . . J., 1:792, 1979) and discold lupus erythematosus (Knop et al., Arch. Dermatol Res., 271:165-70, 1981). In these studies, dosages of **thalidomide** ranging from 100 mg/day to 800 mg/day were administered without serious side effects.

SUMM A further objective of the present invention is the treatment of dermatoses with an autoimmune and/or inflammatory component with **thalidomide** alone or in combination with other agents that inhibit cytokines and/or growth factors, and/or with other classes of therapeutics used. . .

SUMM Another objective of the present invention is the use of **thalidomide** alone or in combination with other agents.

SUMM . . . objective of the current invention is to provide a method for treating dermatoses with an autoimmune and/or inflammatory component with **thalidomide** at a given regimen.

SUMM A further objective of the present invention is a method for the treatment of dermatoses which comprises therapy with **thalidomide** and other drugs on alternative days by diverse schedules.

SUMM An additional objective of the current invention is to utilize **thalidomide** alone or in combination with other inhibitors of cytokines and/or growth factors and/or other treatments for dermatoses

as a maintenance. . .

SUMM A still further objective of this invention is to use **thalidomide** alone or in combination with other inhibitors of cytokines and/or growth factors and/or other treatments for dermatoses as a prophylactic. . .

SUMM . . . dermatoses in a mammal which comprises applying and/or administering to said mammal a composition comprising: (a) an effective amount of **thalidomide** and (b) a therapeutically-acceptable vehicle for the **thalidomide**.

SUMM . . . selected from the group consisting of TNF-alpha inhibitors, basic fibroblast growth factor inhibitors and IL-1 beta inhibitors. Typical inhibitors include **thalidomide** and pentoxifylline but the invention is not limited to those.

SUMM The following is a list of examples of dermatological conditions for which **thalidomide** therapy as proposed in this application is useful. However, proposed **thalidomide** treatments will not be limited to these indications since there may be other dermatological conditions not mentioned here where **thalidomide** may also be effective as a therapeutic:

SUMM (r) Diseases of Mucous Membranes: such as **aphthous** ulcers.

SUMM In treating Kaposi's Sarcoma, an ointment containing 10% by weight of **thalidomide** is applied to the lesion. In an alternative embodiment, Kaposi's Sarcoma is treated concurrently by topical and oral treatment. For. . . presenting with Kaposi's Sarcoma is treated daily for two to four weeks with a dosage amount of 50 mg of **thalidomide** a day while an ointment containing 10% by weight **thalidomide** is applied to the lesion three times a day for two to four weeks.

SUMM When used alone, the topically effective amounts of **thalidomide** are typically 5 to 15% by weight in an ointment and is applied one to three times a day for. . .

SUMM Under certain circumstances, it is desirable to administer **thalidomide** therapy simultaneously with other dermatological active agents. For example, a cream containing 5% by weight of **thalidomide** can be administered three times a day while the patient is being given a topical treatment with 1% hydrocortisone. Concurrent administration of oral **thalidomide** with topical **thalidomide** is also a desirable therapeutic goal.

SUMM Additionally, applicants propose to use **thalidomide** alone or in combination with other inhibitors of cytokines and/or growth factors to treat dermatoses. An example of such a combination therapy utilizes **thalidomide** given with pentoxifylline and a glucocorticoid such as dexamethasone. The activity of each of these agents would be expected to. . . these agents acts as an inhibitor at a different point in this synthesis. Pentoxifylline inhibits TNF alpha gene transcription, while **thalidomide** enhances TNF alpha m-RNA degradation and glucocorticoids, such as dexamethasone, inhibit TNF alpha m-RNA translation.

SUMM The precise amount of **thalidomide** used alone or with other dermatologic agents varies depending, for example, on the condition for which the drug is administered and the size and kind of the mammal. Generally speaking the **thalidomide** can be employed in any amount effective in the treatment of dermatoses.

SUMM For humans, typically-effective amounts of **thalidomide** for use in the topical dosage forms compositions of the present invention range from 5-15% by weight active, however, greater. . .

SUMM . . . be obvious to those skilled in the art that the following

dosage forms may comprise as the active component either **thalidomide** alone or in combination with other compounds. Preferably the compounds of the present invention are administered orally, intramuscularly, topically, subcutaneously,. . .

SUMM It is also possible to administer **thalidomide** in a time-release formulation. A wide variety of methods are now available in the art for preparing time-release or long-acting. . . suitable in the practice of the present invention as long as it does not adversely affect the effectiveness of the **thalidomide** in the treatment of dermatoses. Advantages of time-release formulations include a lower concentration of peak serum absorption which substantially reduces. . . A frequency of administration of every 12 or 24 hours would be preferred. In addition, more constant serum concentration of **thalidomide** would result thereby allowing a more consistent relief of symptoms.

DETD

	Quantity (mg/capsules)
Thalidomide	250
Starch dried	200
Magnesium stearate	10

DETD

	Quantity (mg/tablet)
Thalidomide	250
Cellulose, microcrystalline	400
Silicon dioxide, fumed	10
Stearic acid	5

DETD

Thalidomide	60	mg
Starch	45	mg
Microcrystalline cellulose	35	mg
Polyvinylpyrrolidone (as 10% solution in water)	4	mg
Sodium carboxymethyl starch	4.5	mg
Magnesium stearate	0.5	mg
Talc. . .		

DETD

Thalidomide	80	mg
Starch	59	mg
Microcrystalline cellulose	59	mg
Magnesium stearate	2	mg
Total	200	mg

DETD

Thalidomide	150	mg
Starch	164	mg
Microcrystalline cellulose	164	mg
Magnesium stearate	22	mg

Total	500	mg

DETD A topical ointment containing **thalidomide** is prepared as follows:

DETD

	% by weight
Thalidomide	20%
Vegetable oil	10%
Acetyl lanolin	10%
Lanolin alcohol	12%
Sorbitol sesquioleate	20%
Water add to	100%

DETD

	% by weight
Thalidomide	15%
Carboxyvinyl polymers	2%
Preservative	0.01%
Water add to	100%

DETD

Thalidomide	6.0	g
Stearyl alcohol	3.0	g
Lanolin	5.0	g
Vaseline	15.0	g
d H.sub.2 O added to	100.0	g

DETD Liposomes containing **thalidomide** are made as follows:

DETD Ointment containing **thalidomide**:

DETD

Thalidomide	0.9	g
Hydrocortisone	0.1	g
Isopropyl myristate	81.7	g
Liquid petrolatum oil	9.1	g
Silica - aerosil 200	9.18	g

DETD Twenty patients suffering from psoriasis are to be treated with a cream containing 8% by weight of **thalidomide**.

DETD . . . commercially available product. This commercially available product should be designated the "control", whereas the cream containing 8% by weight of **thalidomide** should be the "test" cream.

DETD These data will clearly demonstrate that the therapeutic composition according to the invention containing 8% by weight **thalidomide** is efficacious and, furthermore, is preferred by the patient to a widely used commercially-available pharmaceutical preparation.

DETD Forty patients suffering from moderate acne are to be treated with a cream containing 5% by weight **thalidomide**.

DETD . . . of the pharmaceutical composition according to the invention, the clinical study should compare this composition with an appropriate placebo (without **thalidomide**) and another commercially available product specifically prescribed for the treatment of acne.

DETD Upon completion of the treatment period, the areas treated with the 5% by weight **thalidomide** cream will exhibit a clinically

significant decrease in the severity of acne as compared to placebo treatment. Furthermore, the **thalidomide**-treated subjects will exhibit less severe side effects and complaints as compared to some other commercially available treatments.

DETD . . . exhibiting leg lesions and diagnosed as being Kaposi's sarcoma are to be treated with a cream containing 10% by weight **thalidomide**.

DETD . . . of the pharmaceutical composition according to the invention, the clinical study should compare this composition with an appropriate placebo (without **thalidomide**) and another commercially available product specifically prescribed for the treatment of Kaposi's sarcoma.

DETD . . . Example 13, two patients are treated except that concurrently with topical administration they are orally treated with 50 mg/day of **thalidomide** for the duration of the topical treatment.

CLM What is claimed is:

. . . mammal which comprises administering to said mammal a therapeutically effective amount of a composition comprising: (a) an effective amount of **thalidomide** and (b) a therapeutically acceptable vehicle for **thalidomide**.

12. The method of claim 11 wherein said TNF alpha inhibitor is selected from the group consisting of **thalidomide** and pentoxifylline.

. . . applying to involved areas of the body and/or administering to said mammal a composition comprising: (a) an effective amount of **thalidomide** and; (b) a therapeutically-acceptable vehicle for the **thalidomide**.

. . . 14. A dermatological composition suitable for treating inflammatory and autoimmune dermatoses in a mammal comprising: a) an effective amount of **thalidomide**; (b) an effective amount of an addition dermatologic drug selected from one group consisting of menthol, phenol, camphor, coal tar. . .

IT 50-23-7, Hydrocortisone **50-35-1**, Thalidomide 53-06-5, Cortisone 57-62-5, Aureomycin 69-72-7, Salicylic acid, biological studies 76-22-2, Camphor 89-78-1, Menthol 108-46-3, Resorcinol, biological studies 108-95-2, Phenol, biological studies 130-26-7, Vioform 1314-13-2, Zinc oxide, biological studies 1404-04-2, Neomycin 1405-41-0, Garamycin **6493-05-6** 7439-97-6D, Mercury, ammoniated, biological studies 7704-34-9, Sulfur, biological studies 65454-29-7, Chloromycin
(pharmaceutical compns. contg. thalidomide for treatment of inflammatory and/or autoimmune dermatoses)

L14 ANSWER 8 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AB Tumour necrosis factor-.alpha. (TNF-.alpha.) is a pleiotropic molecule produced in response to a variety of stimuli during normal host defence. At low levels, TNF-.alpha. confers protection against infectious agents, tumours and tissue damage, and plays a role in the development of humoral immunity. However, overproduction of TNF-.alpha. has been implicated in the pathogenesis of a wide variety of conditions, including autoimmunity, malignancy, inflammatory and immunopathological diseases. Furthermore, TNF-.alpha. is a key regulator of other pro-inflammatory cytokines; infiltrating mononuclear cells that produce excessive amounts of

TNF-.alpha. at sites of inflammation are, therefore, primary targets for therapeutic intervention. Traditional anti-inflammatory drugs, such as **cyclosporin**, have widespread immunosuppressive effects and are now being replaced by more specific anti-TNF-.alpha. compounds [1]. In this report, work presented at the recent Cambridge Symposia meeting on TNF-.alpha. antagonists in Santa Fe, New Mexico, will be highlighted and discussed.

AN 97239656 EMBASE
DN 1997239656
TI Biologicals and Immunologicals. TNF-.alpha. antagonists: Monoclonal antibodies, soluble receptors, **thalidomide** and other novel approaches.
AU Marriott J.B.
CS J.B. Marriott, Division of Oncology, Dept. Cellular / Molecular Sciences, St.George's Hospital Medical School, Cranmer Terrace, London SW17 0RE, United Kingdom
SO Expert Opinion on Investigational Drugs, (1997) 6/8 (1105-1108).
Refs: 17
ISSN: 1354-3784 CODEN: EOIDER
CY United Kingdom
DT Journal; Conference Article
FS 005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
030 Pharmacology
031 Arthritis and Rheumatism
037 Drug Literature Index
048 Gastroenterology
LA English
SL English
TI Biologicals and Immunologicals. TNF-.alpha. antagonists: Monoclonal antibodies, soluble receptors, **thalidomide** and other novel approaches.
SO Expert Opinion on Investigational Drugs, (1997) 6/8 (1105-1108).
Refs: 17
ISSN: 1354-3784 CODEN: EOIDER
AB . . . excessive amounts of TNF-.alpha. at sites of inflammation are, therefore, primary targets for therapeutic intervention. Traditional anti-inflammatory drugs, such as **cyclosporin**, have widespread immunosuppressive effects and are now being replaced by more specific anti-TNF-.alpha. compounds [1]. In this report, work presented. . .
CT Medical Descriptors:
*immunopathology: ET, etiology
*inflammatory disease: ET, etiology
animal model
aphthous ulcer: DT, drug therapy
autoimmunity
clinical trial
conference paper
crohn disease: DT, drug therapy
host resistance
human
humoral immunity
infection
malignant neoplastic disease: ET, etiology
mononuclear cell
nonhuman
protection
rheumatoid arthritis: DT, drug. . .
factor alpha: EC, endogenous compound
*tumor necrosis factor alpha antagonist: PD, pharmacology

*tumor necrosis factor alpha antagonist: DV, drug development
antiinflammatory agent: PD, pharmacology
cyclosporin: PD, pharmacology
cytokine: EC, endogenous compound
immunosuppressive agent: PD, pharmacology
monoclonal antibody: DV, drug development
monoclonal antibody: PD, pharmacology
monoclonal antibody ca2: DT, drug therapy
monoclonal antibody ca2: DV, drug development
monoclonal antibody ca2: CT, clinical trial
monoclonal antibody ca2: PD, pharmacology
thalidomide: PD, pharmacology
thalidomide: CT, clinical trial
tumor necrosis factor alpha antibody: PD, pharmacology
tumor necrosis factor alpha antibody: DV, drug development
tumor necrosis factor receptor: EC,. . .
RN (**cyclosporin**) 79217-60-0; (**thalidomide**) 50-35-1

L14 ANSWER 9 OF 31 CAPLUS COPYRIGHT 2002 ACS
AB **Thalidomide** is very effective in the treatment of idiopathic **aphthous** stomatitis, characterized by recurrent focal intramucosal leukocytic vasculitis. The mode of action of **thalidomide** in this clin. entity may include inhibition of the extravasation of leukocytes. Therefore, the effect of **thalidomide** was studied on different steps of leukocyte migration by intravital microscopy. Leukocyte migration in buccal mucosa of the hamster cheek pouch was elicited by the local application of lipopolysaccharide (LPS, 20 .mu.g/mL) or murine tumor necrosis factor-.alpha. (muTNF-.alpha., 10 ng/mL). (+)-**Thalidomide** (20-200 mg/ kg i.p.) was administered 60 min before the local application of LPS or muTNF-.alpha.. Dexamethasone (2 .times. 1.0-10 mg/kg i.p.) was administered 18 h and 60 min before topical LPS application. The nos. of rolling, firmly adherent, and migrating leukocytes were estd. by intravital microscopy up to 165 min after the topical applications of LPS or muTNF-.alpha. and evaluated by an interactive image anal. software. **Thalidomide** (20-200 mg/kg i.p.) dose-dependently inhibited LPS-stimulated perivenular leukocyte migration by 87% and mu TNF-.alpha.-induced leukocyte migration by 78%. Dexamethasone (2 .times. 1.0-10 mg/kg i.p.) inhibited LPS-stimulated leukocyte migration by 85%. (+)-**Thalidomide** (200 mg/kg i.p.) inhibited LPS-stimulated rolling by 80% and reduced the no. of firmly adherent leukocytes by about 40%. Dexamethasone (2 .times. 10 mg/kg i.p.) did not reduce the no. of rolling leukocytes but inhibited leukocyte adherence by 72%. These results show that (+)-**thalidomide** predominantly inhibits leukocyte rolling and thus differs from the glucocorticoid dexamethasone. The inhibition of LPS- or mu TNF-.alpha.-induced leukocyte extravasation by **thalidomide** may account for some of its clin. activities.
AN 1998:66340 CAPLUS
DN 128:70527
TI Extravasation of leukocytes assessed by intravital microscopy. Effect of **thalidomide**
AU Schneider, J.; Bruckmann, W.; Zwingenberger, K.
CS Gruenenthal G.m.b.H., Aachen, D-52078, Germany
SO Inflammation Research (**1997**), 46(10), 392-397
CODEN: INREFB; ISSN: 1023-3830
PB Birkhaeuser Verlag
DT Journal
LA English

TI Extravasation of leukocytes assessed by intravital microscopy. Effect of **thalidomide**
SO Inflammation Research (**1997**), 46(10), 392-397
CODEN: INREFB; ISSN: 1023-3830
AB **Thalidomide** is very effective in the treatment of idiopathic **aphthous** stomatitis, characterized by recurrent focal intramucosal leukocytic vasculitis. The mode of action of **thalidomide** in this clin. entity may include inhibition of the extravasation of leukocytes. Therefore, the effect of **thalidomide** was studied on different steps of leukocyte migration by intravital microscopy. Leukocyte migration in buccal mucosa of the hamster cheek pouch was elicited by the local application of lipopolysaccharide (LPS, 20 .mu.g/mL) or murine tumor necrosis factor-.alpha. (muTNF-.alpha., 10 ng/mL). (+)-**Thalidomide** (20-200 mg/ kg i.p.) was administered 60 min before the local application of LPS or muTNF-.alpha.. Dexamethasone (2 .times. 1.0-10 mg/kg i.p.) was administered 18 h and 60 min before topical LPS application. The nos. of rolling, firmly adherent, and migrating leukocytes were estd. by intravital microscopy up to 165 min after the topical applications of LPS or muTNF-.alpha. and evaluated by an interactive image anal. software. **Thalidomide** (20-200 mg/kg i.p.) dose-dependently inhibited LPS-stimulated perivenular leukocyte migration by 87% and mu TNF-.alpha.-induced leukocyte migration by 78%. Dexamethasone (2 .times. 1.0-10 mg/kg i.p.) inhibited LPS-stimulated leukocyte migration by 85%. (+)-**Thalidomide** (200 mg/kg i.p.) inhibited LPS-stimulated rolling by 80% and reduced the no. of firmly adherent leukocytes by about 40%. Dexamethasone (2 .times. 10 mg/kg i.p.) did not reduce the no. of rolling leukocytes but inhibited leukocyte adherence by 72%. These results show that (+)-**thalidomide** predominantly inhibits leukocyte rolling and thus differs from the glucocorticoid dexamethasone. The inhibition of LPS- or mu TNF-.alpha.-induced leukocyte extravasation by **thalidomide** may account for some of its clin. activities.
ST **thalidomide** leukocyte migration immunomodulator dexamethasone
IT Immunomodulators
Leukocyte
(extravasation of leukocytes, effect of **thalidomide**)
IT Cell migration
(leukocyte; extravasation of leukocytes, effect of **thalidomide**)
IT Leukocyte
(migration; extravasation of leukocytes, effect of **thalidomide**)
IT **50-02-2**, Dexamethasone 2614-06-4, (+)-**Thalidomide**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(extravasation of leukocytes, effect of)

L14 ANSWER 10 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 97222368 EMBASE
DN 1997222368
TI [Advices in **aphthous** stomatitis].
ADVIEZEN BIJ AFTEN.
AU Wielink G.
CS G. Wielink, Hofstraat 16, 7121 DM Aalten, Netherlands
SO Huisarts en Wetenschap, (1997) 40/8 (389-395).

Refs: 73
ISSN: 0018-7070 CODEN: HUWEAZ
CY Netherlands
DT Journal; (Short Survey)
FS 004 Microbiology
011 Otorhinolaryngology
013 Dermatology and Venereology
037 Drug Literature Index
LA Dutch
SL Dutch
TI [Advices in **aphthous** stomatitis].
ADVIEZEN BIJ AFTEN.
SO Huisarts en Wetenschap, (1997) 40/8 (389-395).
Refs: 73
ISSN: 0018-7070 CODEN: HUWEAZ
CT Medical Descriptors:
***aphthous stomatitis: ET, etiology**
***aphthous stomatitis: TH, therapy**
***aphthous stomatitis: DT, drug therapy**
***aphthous stomatitis: CO, complication**
***aphthous stomatitis: EP, epidemiology**
***aphthous stomatitis: DI, diagnosis**
herpes
human
human immunodeficiency virus infection
literature
morbidity
oral drug administration
recurrent disease
short survey
topical drug administration
*corticosteroid: DT, drug therapy
*lidocaine: DT, drug therapy
*lidocaine: PR, pharmaceutics
*silver nitrate: DT, drug therapy
***thalidomide: DT, drug therapy**
*toothpaste: DT, drug therapy
*toothpaste: PR, pharmaceutics
amlexanox: DT, drug therapy
benzoic acid: PR, pharmaceutics
benzoic acid: DT, drug therapy
betamethasone: DT, drug therapy
chlorhexidine: PR, pharmaceutics
chlorhexidine: DT, drug therapy
fluocinonide: DT, drug therapy
hexetidine: DT, drug therapy
levamisole: DT, drug therapy
mesalazine: DT, drug therapy
prostaglandin e2: DT, drug therapy
sucralfate: DT, drug therapy
triamcinolone: DT,. . .
RN (lidocaine) 137-58-6, 24847-67-4, 56934-02-2, 73-78-9; (silver nitrate) 7761-88-8; (**thalidomide**) 50-35-1; (amlexanox) 68302-57-8; (benzoic acid) 532-32-1, 582-25-2, 65-85-0, 766-76-7; (betamethasone) 378-44-9; (chlorhexidine) 3697-42-5, 55-56-1; (**fluocinonide**) 356-12-7; (hexetidine) 141-94-6; (levamisole) 14769-73-4, 16595-80-5; (mesalazine) 89-57-6; (prostaglandin e2) 363-24-6; (sucralfate) 54182-58-0; (triamcinolone) 124-94-7

L14 ANSWER 11 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AB The cause of recurrent **aphthous** ulcers (RAU), the lesions of recurrent **aphthous** stomatitis, is incompletely understood but appears to involve immune system dysfunction. Treatment options include no
treatment, treatment of associated systemic diseases or conditions (eg, celiac sprue, vitamin deficiencies), systemic medications, topical medications, conversion of the **aphthous** ulcer to a wound, and palliative treatments. The most effective treatments (systemic or topical corticosteroids, **thalidomide**) involve agents that suppress or modulate immune system function. In general, topical agents are preferred because they have fewer associated side effects; however, inability to obtain adequate contact time may limit their effectiveness. Adjunct pain control is sometimes necessary ether with pain medications or with adherent agents that coat the ulcers.

AN 1998004823 EMBASE

TI Topical and systemic therapy for recurrent **aphthous** stomatitis.

AU MacPhail L.
CS Dr. L. MacPhail, UCSF, Department of Stomatology, Box 0422, 513 Parnassus,
San Francisco, CA 94143-0422, United States

SO Seminars in Cutaneous Medicine and Surgery, (1997) 16/4 (301-307).
Refs: 78
ISSN: 1085-5629 CODEN: SCMSFR
CY United States
DT Journal; Conference Article
FS 011 Otorhinolaryngology
037 Drug Literature Index
LA English
SL English

TI Topical and systemic therapy for recurrent **aphthous** stomatitis.
SO Seminars in Cutaneous Medicine and Surgery, (1997) 16/4 (301-307).
Refs: 78
ISSN: 1085-5629 CODEN: SCMSFR
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CT Medical Descriptors:
***aphthous stomatitis: DT, drug therapy**
recurrent disease
drug efficacy
herpes simplex virus
mouth ulcer
immune response
cell adhesion
disease severity
macrophage activation
human
oral drug administration
topical drug administration
conference paper
*aciclovir: AD, drug administration
*aciclovir: DO, drug dose

*aciclovir: DT, drug therapy
*aciclovir: PD, pharmacology
***thalidomide: AD, drug administration**
***thalidomide: DO, drug dose**
***thalidomide: DT, drug therapy**
***thalidomide: PD, pharmacology**
fluocinonide: AD, drug administration
fluocinonide: DO, drug dose
fluocinonide: DT, drug therapy
fluocinonide: PD, pharmacology
clobetasol propionate: AD, drug administration
clobetasol propionate: DO, drug dose
clobetasol propionate: DT, drug therapy
clobetasol propionate: PD, pharmacology
ulobetasol propionate: AD, drug. . .
RN (aciclovir) 59277-89-3; (**thalidomide**) 50-35-1; (**fluocinonide**) 356-12-7; (clobetasol propionate) 25122-46-7; (ulobetasol propionate) 66852-54-8; (dexamethasone) 50-02-2; (clotrimazole) 23593-75-1; (amlexanox) 68302-57-8
CN (1) Zovirax; Lidex; Temovate; Ultravate; **Decadron**; Zilactin; Aphthasol

L14 ANSWER 12 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB **Oral lesions** cause considerable morbidity in association with HIV infection. Their successful management depends upon accurate diagnosis and the use of appropriate therapy. Various treatment approaches are described for some of the common **oral lesions** including Kaposi's sarcoma, oral candidiasis, hairy leukoplakia and recurrent **oral ulcers** associated with HIV disease. This paper will discuss the therapies available in the USA and UK. In other countries some of the drugs discussed will be available in different doses and preparations. In addition other drugs may be available in other parts of the world that are not licensed for use in the USA or UK, and their availability may vary.
AN 97171974 EMBASE
DN 1997171974
TI Management of the oral mucosal lesions seen in association with HIV infection.
AU Greenspan D.; Shirlaw P.J.
CS D. Greenspan, Dept. of Stomatology, Univ. of California San Francisco, 513 Parnassus Avenue, San Francisco, CA 94143-0422, United States
SO Oral Diseases, (1997) 3/SUPPL. 1 (S229-S234).
Refs: 32
ISSN: 1354-523X CODEN: ORDIFD
CY United Kingdom
DT Journal; Conference Article
FS 004 Microbiology
011 Otorhinolaryngology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
SO Oral Diseases, (1997) 3/SUPPL. 1 (S229-S234).
Refs: 32
ISSN: 1354-523X CODEN: ORDIFD
AB **Oral lesions** cause considerable morbidity in association with HIV infection. Their successful management depends upon

accurate diagnosis and the use of appropriate therapy. Various treatment approaches are described for some of the common **oral lesions** including Kaposi's sarcoma, oral candidiasis, hairy leukoplakia and recurrent **oral ulcers** associated with HIV disease. This paper will discuss the therapies available in the USA and UK. In other countries some. . .

CT Medical Descriptors:
*human . . .
therapy
clobetasol propionate: DT, drug therapy
clotrimazole: DT, drug therapy
dexamethasone: DT, drug therapy
fluconazole: DT, drug therapy
fluconazole: AE, adverse drug reaction
fluconazole: IT, drug interaction
fluocinonide: DT, drug therapy
itraconazole: DT, drug therapy
itraconazole: AE, adverse drug reaction
itraconazole: IT, drug interaction
ketoconazole: DT, drug therapy
ketoconazole: IT, drug interaction
ketoconazole: AE,. . . drug therapy
retinoic acid: DT, drug therapy
sclerosing agent: DT, drug therapy
terfenadine: IT, drug interaction
tetracycline: DT, drug therapy
tetradecyl sulfate sodium: DT, drug therapy
thalidomide: DT, drug therapy
triamcinolone acetonide: DT, drug therapy
ulobetasol propionate: DT, drug therapy
unindexed drug
valaciclovir: DT, drug therapy
vinblastine sulfate: DT, drug therapy

RN. . . 378-44-9; (carboxymethylcellulose) 8050-38-2, 9000-11-7, 9004-32-4, 9050-04-8; (chlorhexidine gluconate) 18472-51-0; (clindamycin) 18323-44-9; (clobetasol propionate) 25122-46-7; (clotrimazole) 23593-75-1; (dexamethasone) 50-02-2; (fluconazole) 86386-73-4; (**fluocinonide**) 356-12-7; (itraconazole) 84625-61-6; (ketoconazole) 65277-42-1; (metronidazole) 39322-38-8, 443-48-1; (miconazole) 22916-47-8; (nystatin) 1400-61-9, 34786-70-4, 62997-67-5; (podophyllin) 9000-55-9; (retinoic acid) 302-79-4; (terfenadine) 50679-08-8; (tetracycline) 23843-90-5, 60-54-8, 64-75-5; (tetradecyl sulfate sodium) 1191-50-0, 139-88-8, 4754-44-3; (**thalidomide**) 50-35-1; (triamcinolone acetonide) 76-25-5; (ulobetasol propionate) 66852-54-8; (valaciclovir) 124832-26-4; (vinblastine sulfate) 143-67-9

CN Fungilin; Mycostatin; Mycelex; Daktarin; Corsodyl; Nizoral; Hismanal; Seldane; Diflucan; Sporanox; Zovirax; Valtrex; Retin a; Augmentin; Flagyl; Lidex; Temovate; Ultravate; **Decadron**

L14 ANSWER 13 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 96080664 EMBASE
DN 1996080664
TI Oral manifestations of pediatric human immunodeficiency virus infection: A review of the literature.
AU Kline M.W.
CS Department of Pediatrics, Baylor College of Medicine, One Baylor Plaza,Houston, TX 77030, United States

SO Pediatrics, (1996) 97/3 (380-388).
ISSN: 0031-4005 CODEN: PEDIAU
CY United States
DT Journal; General Review
FS 004 Microbiology
007 Pediatrics and Pediatric Surgery
011 Otorhinolaryngology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SO Pediatrics, (1996) 97/3 (380-388).
ISSN: 0031-4005 CODEN: PEDIAU
CT Medical Descriptors:
***aphthous ulcer: CO, complication**
***aphthous ulcer: SI, side effect**
***aphthous ulcer: PC, prevention**
***aphthous ulcer: DT, drug therapy**
***aphthous ulcer: DI, diagnosis**
*dental caries: DI, diagnosis
*dental caries: CO, complication
*dental caries: PC, prevention
*human immunodeficiency virus infection: DI, diagnosis
*human immunodeficiency virus infection:. . .
AD, drug administration
corticosteroid: AD, drug administration
corticosteroid: DO, drug dose
corticosteroid: DT, drug therapy
fluconazole: DT, drug therapy
fluconazole: DO, drug dose
fluconazole: AD, drug administration
fluocinonide: AD, drug administration
fluocinonide: PR, pharmaceutics
fluocinonide: DT, drug therapy
hydrocortisone: PR, pharmaceutics
hydrocortisone: DT, drug therapy
hydrocortisone: AD, drug administration
hydrocortisone: CB, drug combination
lidocaine: PR, pharmaceutics
lidocaine: CB, drug combination
lidocaine: AD,. . . therapy
prednisone: DT, drug therapy
prednisone: DO, drug dose
prednisone: AD, drug administration
tetracycline: DT, drug therapy
tetracycline: PR, pharmaceutics
tetracycline: AD, drug administration
tetracycline: CB, drug combination
thalidomide: DO, drug dose
thalidomide: DT, drug therapy
RN (zalcitabine) 7481-89-2; (ganciclovir) 82410-32-0; (ketoconazole)
65277-42-1; (zidovudine) 30516-87-1; (aciclovir) 59277-89-3;
(bethanechol)
590-63-6, 674-38-4, 91609-06-2; (clotrimazole) 23593-75-1; (fluconazole)
86386-73-4; (**fluocinonide**) 356-12-7; (hydrocortisone) 50-23-7;
(lidocaine) 137-58-6, 24847-67-4, 56934-02-2, 73-78-9; (nystatin)
1400-61-9, 34786-70-4, 62997-67-5; (prednisone) 53-03-2; (tetracycline)
23843-90-5, 60-54-8, 64-75-5; (**thalidomide**) 50-35-1

L14 ANSWER 14 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB We review the cutaneous manifestations of acute and chronic graft versus

host disease (GvHD). Acute GvHD is characterized by initial itching, pain on pressure and erythema which begins on posterior auricular skin, palms and soles. The disease evolves into a typical but nonspecific maculopapular rash. Confluent rashes and follicular erythema may occur. Erosive **oral lesions** usually develop. The most severe variant of GvHD is toxic epidermal necrolysis, which often has a fatal outcome. The onset of chronic GvHD usually occurs more than 100 days after bone marrow transplantation and may be preceded by the acute form. The spectrum of skin changes includes lichenoid pruritic lesions with violaceous color and scleroderma-like skin involvement. Investigation of unknown rashes in these patients includes skin biopsy, which clearly differentiates leukocytoclastic vasculitis and erythema exsudativum multiforme with lymphocytic vasculitis from cutaneous manifestations of GvHD. Special stains may reveal bacteria and fungus in septicemic patients. The therapeutic options are discussed.

AN 96074372 EMBASE
DN 1996074372
TI [Cutaneous manifestations of graft versus host disease after bone marrow transplantation].
HAUTMANIFESTATIONEN DER GRAFT-VERSUS-HOST-REAKTION NACH KNOCHENMARKTRANSPLANTATION.
AU Itin P.H.; Lautenschlager S.; Orth B.; Rufli T.; Gratwohl A.
CS Dermatologische Universitatsklinik, Petersgraben 4,CH-4031 Basel, Switzerland
SO Schweizerische Medizinische Wochenschrift, (1996) 126/9 (339-347). ISSN: 0036-7672 CODEN: SMWOAS
CY Switzerland
DT Journal; General Review
FS 013 Dermatology and Venereology
025 Hematology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA German
SL German; English
SO Schweizerische Medizinische Wochenschrift, (1996) 126/9 (339-347). ISSN: 0036-7672 CODEN: SMWOAS
AB . . . and soles. The disease evolves into a typical but nonspecific maculopapular rash. Confluent rashes and follicular erythema may occur. Erosive **oral lesions** usually develop. The most severe variant of GvHD is toxic epidermal necrolysis, which often has a fatal outcome. The onset. . .
CT Medical Descriptors:
*bone . . . host reaction: CO, complication
human
infection: PC, prevention
infection: DT, drug therapy
infection: CO, complication
puva
review
skin manifestation: ET, etiology
corticosteroid: DT, drug therapy
corticosteroid: CB, drug combination
cyclosporin a: DT, drug therapy
cyclosporin a: CB, drug combination
lymphocyte antibody: DT, drug therapy
methotrexate: DT, drug therapy
methotrexate: CB, drug combination
povidone iodine: DT, drug therapy
thalidomide: DT, drug therapy

tumor necrosis factor antibody: DT, drug therapy
RN (**cyclosporin** a) 59865-13-3, 63798-73-2; (methotrexate) 15475-56-6, 59-05-2, 7413-34-5; (povidone iodine) 25655-41-8; (**thalidomide**) 50-35-1; (tumor necrosis factor antibody) 162774-06-3

L14 ANSWER 15 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB **Oral lesions** are common in HIV infection and may be the first sign of AISDS. this article reviews the oral fungal and viral infections commonly detected in HIV-infected patients, particularly candidiasis, deep fungal infections, herpes simplex virus infections, cytomegalovirus infections, and oral hairy leukoplakia. The neoplasms associated with AIDS such as oral Kaposi's sarcoma and lymphoma are related periodontal diseases. Each disorder is discussed by clinical appearance, diagnosis, and management. Recent advances in therapy are stressed.
AN 96149440 EMBASE
DN 1996149440
TI HIV-associated lesions.
AU Greenberg M.S.
CS Departmentof Oral Medicine, School of Dental Medicine, University of Pennsylvania, 4001 Spruce Street,Philadelphia, PA 19104-6003, United States
SO Dermatologic Clinics, (1996) 14/2 (319-326).
ISSN: 0733-8635 CODEN: DRMCDJ
CY United States
DT Journal; General Review
FS 004 Microbiology
011 Otorhinolaryngology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
SO Dermatologic Clinics, (1996) 14/2 (319-326).
ISSN: 0733-8635 CODEN: DRMCDJ
AB **Oral lesions** are common in HIV infection and may be the first sign of AISDS. this article reviews the oral fungal and. . .

CT Medical Descriptors:
*acquired . . . therapy
*kaposi sarcoma: ET, etiology
*mycosis: ET, etiology
*mycosis: DT, drug therapy
*mycosis: DI, diagnosis
*virus infection: ET, etiology
*virus infection: DT, drug therapy
*virus infection: DI, diagnosis
aphthous ulcer: ET, etiology
aphthous ulcer: DT, drug therapy
aphthous ulcer: DI, diagnosis
candidiasis: DT, drug therapy
candidiasis: DI, diagnosis
candidiasis: CO, complication
cheilitis: DI, diagnosis
cheilitis: ET, etiology
cryptococcosis: DT, drug therapy
cryptococcosis: ET, etiology
cryptococcosis: DI,. . .
drug therapy
amphotericin b: DT, drug therapy

capsaicin: DT, drug therapy
clobetasol: DT, drug therapy
clotrimazole: DT, drug therapy
dapsone: DT, drug therapy
fluconazole: DT, drug therapy
fluocinonide: DT, drug therapy
foscarnet: DT, drug therapy
foscarnet: AE, adverse drug reaction
ganciclovir: DT, drug therapy
ganciclovir: AE, adverse drug reaction
itraconazole: DT, drug therapy
ketoconazole: DT, drug therapy
metronidazole: DT, drug therapy
miconazole: DT, drug therapy
nystatin: DT, drug therapy
podophyllin: DT, drug therapy
retinoid: DT, drug therapy
thalidomide: DT, drug therapy
tricyclic antidepressant agent: DT, drug therapy
RN. . . 139-88-8, 4754-44-3; (vinblastine) 865-21-4; (aciclovir) 59277-89-3;
(amphotericin b) 1397-89-3, 30652-87-0; (capsaicin) 404-86-4; (clobetasol)
25122-41-2; (clotrimazole) 23593-75-1; (dapsone) 80-08-0; (fluconazole)
86386-73-4; (**fluocinonide**) 356-12-7; (foscarnet) 4428-95-9;
(ganciclovir) 82410-32-0; (itraconazole) 84625-61-6; (ketoconazole)
65277-42-1; (metronidazole) 39322-38-8, 443-48-1; (miconazole) 22916-47-8;
(nystatin) 1400-61-9, 34786-70-4, 62997-67-5; (podophyllin) 9000-55-9; (**thalidomide**) 50-35-1

L14 ANSWER 16 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB Recurrent **aphthous** stomatitis (RAS) is the most common oral mucosal disease in North America. In some instances, RAS represents the central feature of the multisystem disease complex Behcet's syndrome. This article reviews the clinical features, contributing etiologic factors, and etiopathogenesis of RAS and Behcet's syndrome and describes therapeutic considerations and strategies essential to management of patients suffering from recurrent mouth ulcers.
AN 96149434 EMBASE
DN 1996149434
TI Recurrent **aphthous** stomatitis.
AU Rees T.D.; Binnie W.H.
CS Baylor College of Dentistry, 3302 Gaston Avenue,Dallas, TX 75246, United States
SO Dermatologic Clinics, (1996) 14/2 (243-256).
ISSN: 0733-8635 CODEN: DRMCDJ
CY United States
DT Journal; General Review
FS 011 Otorhinolaryngology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
TI Recurrent **aphthous** stomatitis.
SO Dermatologic Clinics, (1996) 14/2 (243-256).
ISSN: 0733-8635 CODEN: DRMCDJ
AB Recurrent **aphthous** stomatitis (RAS) is the most common oral

mucosal disease in North America. In some instances, RAS represents the central feature. . .

CT Medical Descriptors:

***aphthous stomatitis: DI, diagnosis**
***aphthous stomatitis: DT, drug therapy**
***aphthous stomatitis: EP, epidemiology**
***aphthous stomatitis: ET, etiology**
***aphthous stomatitis: TH, therapy**
***aphthous ulcer: DI, diagnosis**
***aphthous ulcer: DT, drug therapy**
***aphthous ulcer: EP, epidemiology**
***aphthous ulcer: ET, etiology**
***aphthous ulcer: TH, therapy**
*behcet disease: ET, etiology
*behcet disease: DT, drug therapy
*behcet disease: DI, diagnosis
food allergy: ET, etiology
gluten free diet
heredity
human
injury
mouth hygiene
priority journal
review
side. . .
DT, drug therapy
azathioprine: AE, adverse drug reaction
betamethasone dipropionate: DT, drug therapy
clobetasol: DT, drug therapy
colchicine: AE, adverse drug reaction
colchicine: DT, drug therapy
cyclosporin: DT, drug therapy
cyclosporin: AE, adverse drug reaction
dapsone: DT, drug therapy
fluocinonide: DT, drug therapy
immunomodulating agent
immunostimulating agent
immunosuppressive agent
levamisole: DT, drug therapy
levamisole: AE, adverse drug reaction
mesalazine: DT, drug therapy
methotrexate: DT, drug therapy
nonsteroid antiinflammatory agent: DT, drug therapy
prednisone: DT, drug therapy
prostaglandin e2: DT, drug therapy
sucralfate: DT, drug therapy
superoxide dismutase: DT, drug therapy
thalidomide: AE, adverse drug reaction
thalidomide: DT, drug therapy

RN (chlorhexidine gluconate) 18472-51-0; (listerine) 51273-66-6; (aciclovir) 59277-89-3; (amlexanox) 68302-57-8; (azathioprine) 446-86-6; (betamethasone dipropionate) 5593-20-4; (clobetasol) 25122-41-2; (colchicine) 64-86-8; (**cyclosporin**) 79217-60-0; (dapsone) 80-08-0; (**fluocinonide**) 356-12-7; (levamisole) 14769-73-4, 16595-80-5; (mesalazine) 89-57-6; (methotrexate) 15475-56-6, 59-05-2, 7413-34-5; (prednisone) 53-03-2; (prostaglandin e2) 363-24-6; (sucralfate) 54182-58-0; (superoxide dismutase) 37294-21-6, 9016-01-7, 9054-89-1; (**thalidomide**) 50-35-1

L14 ANSWER 17 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.DUPLICATE 2
AB Objective. The diagnosis and treatment of the mucocutaneous (MC), neuropsychiatric (NP),and renal (RN) manifestations of systemic lupus erythematosus (SLE) remain unsolved issues. To shed light on these issues, a questionnaire was prepared and sent to 153 lupus centres around the world, in order to determine the level of agreement between experts in their approach to these complex aspects of the disease. Methods. The first section of the questionnaire was designed to collect information on the characteristics of the responding lupus centres. The second section was dedicated to MC manifestations, with questions focusing on: (i) the frequency of MC manifestations as a whole and of the single clinical MC entities; (ii) clinical features, outcome and therapy of subacute cutaneous lupus erythematosus (SCLE); (iii) the utility of the lupus band test (LBT); and (iv) the use of various therapeutic protocols to treat MC manifestations. Results. Sixty-one questionnaires from 19 countries were analysed. Out of these, 37 were completed by Departments of Rheumatology, 21 by Departments of Internal Medicine or Clinical Immunology, and 3 by Departments of Nephrology. About 66% of these centres stated that they were currently following more than 100 lupus cases, 95% had an in-patient ward and 82% had their own laboratory. The American College of Rheumatology classification criteria and various scales for disease activity assessment were regularly used by 87% and 57% of centres, respectively. The overall prevalence of MC manifestations was judged to be over 30% by 82% of the respondents (Rs), and over 60% by 36% of the Rs. Among the different MC manifestations, malar rash was reported to be the most frequent (40%), followed by alopecia (24.1%) and **oral ulcers** (18.6%). In reporting the prevalence of each MC manifestation, the Rs showed a low level of agreement, the coefficient of variation (CV) being > 0.75 for all of the manifestations listed with the exception of malar rash (CV = 0.54). Poor agreement among centers was also found for the reported association of various MC manifestations with SCLE (15 different answers), and on the prognostic factors for SCLE (17 different answers). There was agreement on the best procedure (up to 70% of the Rs preferred a non-UV exposed skin area) and on the utility of the LBT (83% using it only for diagnostic purpose). Hydroxychloroquine was the most popular therapeutic protocol, being used by 85% of the Rs for a wide variety of MC manifestations. Among other therapies, azathioprine was used by 59%, dapsone by 41%, and **thalidomide** by 35% of the Rs, all to treat a wide spectrum of MC manifestations. Pulse steroid, **cyclosporin** A and pulse cyclophosphamide were less commonly employed (by 27%, 22% and 13% of the Rs, respectively), and were reserved for the most severe MC manifestations, particularly vasculitis. Conclusion. The present survey indicates that, although most of the participating centres had extensive experience in the management of SLE, their approach to the MC manifestations was not homogeneous, and collaborative studies are clearly needed, particularly to optimise the therapeutic protocols.
AN 97056106 EMBASE
DN 1997056106
TI International survey on the management of patients with SLE. I. General data on the participating centers and the results of a questionnaire regarding mucocutaneous involvement.
AU Vitali C.; Doria A.; Tincani A.; Fabbri P.; Balestrieri G.; Galeazzi M.; Meroni P.L.; Migliorini P.; Neri R.; Tavoni A.; Bombardieri S.

CS Dr. C. Vitali, U.O. di Immunologia Clinica, Istituto di Patologia Medica, Universita di Pisa, Via Roma 67, 56126 Pisa, Italy
SO Clinical and Experimental Rheumatology, (1996) 14/SUPPL. 16 (S17-S22). Refs: 8
ISSN: 0392-856X CODEN: CERHDP
CY Italy
DT Journal; Conference Article
FS 008 Neurology and Neurosurgery
013 Dermatology and Venereology
026 Immunology, Serology and Transplantation
028 Urology and Nephrology
031 Arthritis and Rheumatism
032 Psychiatry
037 Drug Literature Index
LA English
SL English
SO Clinical and Experimental Rheumatology, (1996) 14/SUPPL. 16 (S17-S22). Refs: 8
ISSN: 0392-856X CODEN: CERHDP
AB . . . Among the different MC manifestations, malar rash was reported to be the most frequent (40%), followed by alopecia (24.1%) and **oral ulcers** (18.6%). In reporting the prevalence of each MC manifestation, the Rs showed a low level of agreement, the coefficient of. . . . Rs for a wide variety of MC manifestations. Among other therapies, azathioprine was used by 59%, dapsone by 41%, and **thalidomide** by 35% of the Rs, all to treat a wide spectrum of MC manifestations. Pulse steroid, **cyclosporin** A and pulse cyclophosphamide were less commonly employed (by 27%, 22% and 13% of the Rs, respectively), and were reserved. . .
CT Medical Descriptors:
*lupus . . . erythematosus: DT, drug therapy
*systemic lupus erythematosus: TH, therapy
clinical trial
conference paper
controlled study
human
priority journal
quality of life
questionnaire
*azathioprine: DT, drug therapy
*cyclophosphamide: DT, drug therapy
***cyclosporin a: DT, drug therapy**
*dapsone: DT, drug therapy
*hydroxychloroquine: DT, drug therapy
***thalidomide: DT, drug therapy**
RN (azathioprine) 446-86-6; (cyclophosphamide) 50-18-0; (**cyclosporin** a) 59865-13-3, 63798-73-2; (dapsone) 80-08-0; (hydroxychloroquine) 118-42-3, 525-31-5; (**thalidomide**) 50-35-1

L14 ANSWER 18 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB Behcet's disease (BD) is a multisystemic disease that must be diagnosed by clinical criteria in the absence of any specific laboratory test or biologic marker. International criteria for the diagnosis have been recently revised. We performed a retrospective study of those cases diagnosed of BD in our center between 1992-1993. Age, sex, clinical manifestations (mucocutaneous, ocular and systemic) and histopathologic findings were revised. HLA study and pathergy test were done in each case.

Seven patients were included, four women and three men. Mucocutaneous manifestations were the most prominent finding and oral aphtae were the initial lesions in 85% of cases. Pustular lesions or folliculitis were present in 71% of cases. Ocular involvement was observed in three cases (42%) (one of these was asymptomatic). We did not find association between HLA-B5 and ocular involvement. Systemic manifestaions were present as arthritis (29%) and thrombophlebitis (14%). Pathergy test was only relevant in one case. The most common histopathologic finding was a neutrophilic vascular reaction. BD is in our study more common than expected. It must be due perhaps to the new criteria proposed for the diagnosis where four of the five criteria are based in the same basic mucocutaneous lesion. We recommend to biopsy pustular lesions before including them as a diagnosis criteria and to perform ocular study using fluorescein in all suspect cases to detect abnormalities in asymptomatic patients.

AN 95351726 EMBASE
DN 1995351726
TI [Behcet's disease: Clinical-pathologic revision of seven cases]. ENFERMEDAD DE BEHCET: REVISION CLINICO-PATOLOGICA DE SIETE CASOS.
AU Quecedo Estebanez E.; Gil Mateo M.P.; Febrer Bosch M.I.; Sanchez Carazo J.L.; Martinez Escribano J.; Velasco Pastor M.; Aliaga Boniche A.
CS Servicio de Dermatologia, Hospital General Universitario, Avda. Tres Cruces, s/n,46014 Valencia, Spain
SO Actas Dermo-Sifiliograficas, (1995) 86/11 (581-588).
ISSN: 0001-7310 CODEN: ADSIAZ
CY Spain
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
006 Internal Medicine
012 Ophthalmology
013 Dermatology and Venereology
018 Cardiovascular Diseases and Cardiovascular Surgery
026 Immunology, Serology and Transplantation
031 Arthritis and Rheumatism
037 Drug Literature Index
LA Spanish
SL English; Spanish
SO Actas Dermo-Sifiliograficas, (1995) 86/11 (581-588).
ISSN: 0001-7310 CODEN: ADSIAZ
CT Medical Descriptors:
*behcet disease: DI, diagnosis
*behcet disease: ET, etiology
*behcet disease: DT, drug therapy
adolescent
adult
aphthous ulcer: DI, diagnosis
aphthous ulcer: ET, etiology
arthritis: ET, etiology
arthritis: DI, diagnosis
article
eye injury: DI, diagnosis
eye injury: ET, etiology
female
folliculitis: ET, etiology
folliculitis: DI, diagnosis
histopathology
human
male
neutrophil

skin defect: DI, diagnosis
skin. . . ET, etiology
topical drug administration
*corticosteroid: DT, drug therapy
*immunosuppressive agent: DT, drug therapy
azathioprine: DT, drug therapy
chlorambucil: DT, drug therapy
cyclophosphamide: DT, drug therapy
cyclosporin a: DT, drug therapy
indometacin: DT, drug therapy
thalidomide: DT, drug therapy
triamcinolone acetonide: DT, drug therapy
RN (azathioprine) 446-86-6; (chlorambucil) 305-03-3; (cyclophosphamide) 50-18-0; (**cyclosporin** a) 59865-13-3, 63798-73-2; (indometacin) 53-86-1, 74252-25-8, 7681-54-1; (**thalidomide**) 50-35-1; (triamcinolone acetonide) 76-25-5

L14 ANSWER 19 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB **Thalidomide** has been advocated as the treatment of choice for recalcitrant **aphthae**. We describe the case of patient with HIV infection and extensive **aphthae** whose condition failed to respond to corticosteroids, cyclosporine, and **thalidomide**. The patient's course was complicated by colonic **aphthae**. Rapid and sustained resolution was achieved through treatment with granulocyte colony-stimulating factor, a previously unreported therapeutic option.
AN 95233657 EMBASE
DN 1995233657
TI **Thalidomide**-resistant HIV-associated **aphthae** successfully treated with granulocyte colony-stimulating factor.
AU Manders S.M.; Kostman J.R.; Mendez L.; Russin V.L.
CS 100 Brick Rd.,Marlton, NJ 08053, United States
SO Journal of the American Academy of Dermatology, (1995) 33/2 II (380-382). ISSN: 0190-9622 CODEN: JAADDB
CY United States
DT Journal; Article
FS 004 Microbiology
011 Otorhinolaryngology
013 Dermatology and Venereology
037 Drug Literature Index
048 Gastroenterology
LA English
SL English
TI **Thalidomide**-resistant HIV-associated **aphthae** successfully treated with granulocyte colony-stimulating factor.
SO Journal of the American Academy of Dermatology, (1995) 33/2 II (380-382). ISSN: 0190-9622 CODEN: JAADDB
AB **Thalidomide** has been advocated as the treatment of choice for recalcitrant **aphthae**. We describe the case of patient with HIV infection and extensive **aphthae** whose condition failed to respond to corticosteroids, cyclosporine, and **thalidomide**. The patient's course was complicated by colonic **aphthae**. Rapid and sustained resolution was achieved through treatment with granulocyte colony-stimulating factor, a previously unreported therapeutic option.
CT Medical Descriptors:
***aphthous ulcer: DI, diagnosis**
***aphthous ulcer: DT, drug therapy**
*human immunodeficiency virus infection
abdominal pain
adult
article

case report
colon biopsy
colon ulcer: SU, surgery
colon ulcer: DT, drug therapy
colon ulcer: DI, diagnosis
colonoscopy
drug resistance
human
leukocyte count
oral drug administration
priority journal
recurrent disease
*granulocyte colony stimulating factor: DT, drug therapy
***thalidomide: DO, drug dose**
***thalidomide: DT, drug therapy**
cyclosporin: DO, drug dose
cyclosporin: DT, drug therapy
hydrocortisone
nystatin
prednisone: DO, drug dose
prednisone: DT, drug therapy
tetracycline

RN (**thalidomide**) 50-35-1; (**cyclosporin**) 79217-60-0; (hydrocortisone) 50-23-7; (nystatin) 1400-61-9, 34786-70-4, 62997-67-5; (prednisone) 53-03-2; (tetracycline) 23843-90-5, 60-54-8, 64-75-5

L14 ANSWER 20 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AB Despite its inherent teratogenic risk, **thalidomide** has over the years proven to be of clinical use in a small number of mainly immunological diseases (e.g. erythema nodosum leprosum, Behcet's syndrome and rheumatoid arthritis). The mode of action of **thalidomide** is still poorly understood. Recent research has shown a decrease in tumour necrosis factor-.alpha. (TNF.alpha.) during **thalidomide** treatment in several settings. Others have found altered expression of adhesion molecules. Currently, the most interesting new fields of application are the prevention and treatment of graft-versus-host disease in allogeneic bone marrow transplantation and the treatment of **aphthous** ulceration in HIV-positive patients. Contraceptive measures must be instituted in women receiving **thalidomide**, and careful monitoring for neurological adverse effects is required in all patients.

AN 95188270 EMBASE

DN 1995188270

TI **Thalidomide**: Rationale for renewed use in immunological disorders.

AU Schuler U.; Ehninger G.

CS Medizinische Klinik I, Fetscherstrasse 74,01307 Dresden, Germany

SO Drug Safety, (1995) 12/6 (364-369).
ISSN: 0114-5916 CODEN: DRSAEA

CY New Zealand

DT Journal; General Review

FS 026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles

LA English

SL English

TI **Thalidomide**: Rationale for renewed use in immunological disorders.

SO Drug Safety, (1995) 12/6 (364-369).

ISSN: 0114-5916 CODEN: DRSAEA

AB Despite its inherent teratogenic risk, **thalidomide** has over the years proven to be of clinical use in a small number of mainly immunological diseases (e.g. erythema nodosum leprosum, Behcet's syndrome and rheumatoid arthritis). The mode of action of **thalidomide** is still poorly understood. Recent research has shown a decrease in tumour necrosis factor-.alpha. (TNF.alpha.) during **thalidomide** treatment in several settings. Others have found altered expression of adhesion molecules. Currently, the most interesting new fields of application are the prevention and treatment of graft-versus-host disease in allogeneic bone marrow transplantation and the treatment of **aphthous** ulceration in HIV-positive patients. Contraceptive measures must be instituted in women receiving **thalidomide**, and careful monitoring for neurological adverse effects is required in all patients.

CT Medical Descriptors:
*immunopathology: DT, drug therapy
allogenic bone marrow transplantation
aphthous ulcer: DT, drug therapy
behcet disease: DT, drug therapy
clinical trial
constipation: SI, side effect
contraception
drowsiness: SI, side effect
drug efficacy
drug mechanism
drug monitoring
eosinophilia: SI, side. . . side effect
neurologic disease
nonhuman
priority journal
pruritus: DT, drug therapy
rash: SI, side effect
review
rheumatoid arthritis: DT, drug therapy
risk
teratogenicity
uremia: DT, drug therapy
xerostomia: SI, side effect
***thalidomide: AE, adverse drug reaction**
***thalidomide: CT, clinical trial**
***thalidomide: DT, drug therapy**
***thalidomide: PD, pharmacology**
cell adhesion molecule: EC, endogenous compound
cyclosporin: DT, drug therapy
methotrexate: DT, drug therapy
tumor necrosis factor alpha: EC, endogenous compound

RN (**thalidomide**) 50-35-1; (**cyclosporin**) 79217-60-0; (methotrexate) 15475-56-6, 59-05-2, 7413-34-5

L14 ANSWER 21 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AB Sutton's disease is characterized by giant necrotizing ulcers around minor salivary glands and is of unknown cause. We report a case, review the medical literature, and discuss the treatment of this affliction.

AN 95126138 EMBASE

DN 1995126138

TI Sutton's disease (periadenitis mucosa necrotica recurrens).

AU Laccourreye O.; Durand H.; Fadlallah J.-P.; Brasnu D.; Pages J.-C.; Lowenstein W.

CS Otorhinolaryn.-Head/Neck Surg Dept., University Paris V, Laennec Hospital,Paris, France
SO Annals of Otology, Rhinology and Laryngology, (1995) 104/4 I (301-304). ISSN: 0003-4894 CODEN: AORHA2
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
011 Otorhinolaryngology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
SO Annals of Otology, Rhinology and Laryngology, (1995) 104/4 I (301-304). ISSN: 0003-4894 CODEN: AORHA2
CT Medical Descriptors:
***aphthous stomatitis: DT, drug therapy**
adult
article
case report
human
male
mouth hygiene
mouth ulcer
mutagenicity
pathophysiology
priority journal
teratogenicity: SI, side effect
aciclovir: DT, drug therapy
azathioprine: DT, drug therapy
betamethasone: DT, drug therapy
chlorhexidine: DT, drug therapy
erythromycin: DT, drug therapy
fluocinonide: DT, drug therapy
levamisole: DT, drug therapy
nystatin: DT, drug therapy
tetracycline: DT, drug therapy
thalidomide: AE, adverse drug reaction
triamcinolone acetonide: DT, drug therapy
zinc sulfate: DT, drug therapy
RN (aciclovir) 59277-89-3; (azathioprine) 446-86-6; (betamethasone) 378-44-9;
(chlorhexidine) 3697-42-5, 55-56-1; (erythromycin) 114-07-8, 70536-18-4; (**fluocinonide**) 356-12-7; (levamisole) 14769-73-4, 16595-80-5; (nystatin) 1400-61-9, 34786-70-4, 62997-67-5; (tetracycline) 23843-90-5, 60-54-8, 64-75-5; (**thalidomide**) 50-35-1; (triamcinolone acetonide) 76-25-5; (zinc sulfate) 7733-02-0

L14 ANSWER 22 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB Behcet's syndrome is a multisystem vasculitis of unknown aetiology. Eye involvement, the main cause of morbidity, can lead to blindness in 20% of those affected. Other lesions, ranging from **aphthous** and genital ulceration to sometimes fatal central nervous system involvement, also cause considerable morbidity and, as we have become more recently aware, mortality. The syndrome runs a course of exacerbations and remissions, and usually abates in intensity with the passage of time. Young adult males have the worst prognosis. The main aim of treatment is to prevent irreversible organ damage during-the early, active, phase of the disease. Immunosuppression remains the mainstay of therapy. Azathioprine is able to

suppress most of the manifestations of the syndrome. **Cyclosporin** has a considerably more rapid onset of action, and is particularly useful in the treatment of uveitis. However, the disease usyally flares on cessation of **cyclosporin** treatment. Neither azathioprine nor **cyclosporin** is always effective, and there are patients who continue to do badly even with their combined use. **Thalidomide** is useful in severe oral ulceration and colchicine in erythema nodosum associated with Behcet's syndrome. There is no established remedy for the central nervous system and thrombotic complications of Behcet's syndrome.

AN 95056456 EMBASE
DN 1995056456
TI Behcet's syndrome: How should we treat it?.
AU Yazici H.; Yurdakul S.; Hamuryudan V.
CS Division of Rheumatology, Dept. Med. Cerrahpasa Med. Faculty, Safa Sok 17/4,Kadikoy, 81310 Istanbul, Turkey
SO Clinical Immunotherapeutics, (1995) 3/2 (102-107).
ISSN: 1172-7039 CODEN: CIMMEA
CY New Zealand
DT Journal; General Review
FS 006 Internal Medicine
011 Otorhinolaryngology
012 Ophthalmology
025 Hematology
026 Immunology, Serology and Transplantation
031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
SO Clinical Immunotherapeutics, (1995) 3/2 (102-107).
ISSN: 1172-7039 CODEN: CIMMEA
AB . . . Eye involvement, the main cause of morbidity, can lead to blindness in 20% of those affected. Other lesions, ranging from **aphthous** and genital ulceration to sometimes fatal central nervous system involvement, also cause considerable morbidity and, as we have become more. . . the disease. Immunosuppression remains the mainstay of therapy. Azathioprine is able to suppress most of the manifestations of the syndrome. **Cyclosporin** has a considerably more rapid onset of action, and is particularly useful in the treatment of uveitis. However, the disease usyally flares on cessation of **cyclosporin** treatment. Neither azathioprine nor **cyclosporin** is always effective, and there are patients who continue to do badly even with their combined use. **Thalidomide** is useful in severe oral ulceration and colchicine in erythema nodosum associated with Behcet's syndrome. There is no established remedy. . .
CT Medical Descriptors:
*behcet . . . SI, side effect
priority journal
review
teratogenicity: SI, side effect
thrombosis: PC, prevention
thrombosis: DT, drug therapy
topical drug administration
*azathioprine: CB, drug combination
*azathioprine: DT, drug therapy
***cyclosporin: DT, drug therapy**
***cyclosporin: CM, drug comparison**
***cyclosporin: DO, drug dose**

***cyclosporin: AE, adverse drug reaction**
***thalidomide: DT, drug therapy**
***thalidomide: AE, adverse drug reaction**
acetylsalicylic acid: DT, drug therapy
acetylsalicylic acid: CB, drug combination
alpha2b interferon: DT, drug therapy
chlorambucil: DT, drug therapy
chlorambucil: AE,. . .
RN (azathioprine) 446-86-6; (**cyclosporin**) 79217-60-0; (**thalidomide**) 50-35-1; (acetylsalicylic acid) 493-53-8, 50-78-2, 53663-74-4, 53664-49-6, 63781-77-1; (alpha2b interferon) 99210-65-8; (chlorambucil) 305-03-3; (colchicine) 64-86-8; (cyclophosphamide) 50-18-0; (methylprednisolone) 6923-42-8, 83-43-2

L14 ANSWER 23 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB Recurrent oral ulcerations is one of the most frequent alterations of the oral mucosa, involving 20% of the general populations. The etiology is unknown, but there are several predisposing factors such as, streptococcal infections, hematinic deficiencies, stress, or certain food. It is known that immunological mechanisms participate in the pathogenesis of the **aphthae**, because of the presence of a lymphocytic inflammatory infiltrate in the early phases of the ulcers. Therapy includes the elimination of the predisposing factors and the topical application of corticosteroids. In severe cases, it may be necessary to administer systemic corticosteroids or of other immunosuppressors. Recurrent oral **aphthous** are one of the criteria of Behcet's syndrome.
AN 95151183 EMBASE
DN 1995151183
TI [Aftosis].
AFTOSIS.
AU Barnadas M.A.
CS Hospital La Santa Cruz y San Pable,Barcelona, Spain
SO Monografias de Dermatologia, (1995) 8/2 (100-110).
ISSN: 0214-4220 CODEN: MONDE4
CY Spain
DT Journal; Article
FS 011 Otorhinolaryngology
012 Ophthalmology
037 Drug Literature Index
LA Spanish
SL Spanish; English
SO Monografias de Dermatologia, (1995) 8/2 (100-110).
ISSN: 0214-4220 CODEN: MONDE4
AB . . . streptococcal infections, hematinic deficiencies, stress, or certain food. It is known that immunological mechanisms participate in the pathogenesis of the **aphthae**, because of the presence of a lymphocytic inflammatory infiltrate in the early phases of the ulcers. Therapy includes the elimination. . . application of corticosteroids. In severe cases, it may be necessary to administer systemic corticosteroids or of other immunosuppressors. Recurrent oral **aphthous** are one of the criteria of Behcet's syndrome.
CT Medical Descriptors:
***aphthous ulcer: DI, diagnosis**
***aphthous ulcer: DT, drug therapy**
***aphthous ulcer: ET, etiology**
*behcet disease
*mouth ulcer: DT, drug therapy

*mouth ulcer: DI, diagnosis
*mouth ulcer: ET, etiology
*uveitis
article
clinical feature
disease predisposition
human
pathogenesis
recurrent disease
topical drug administration
*antibiotic agent: DT, drug therapy
*corticosteroid: DT, drug therapy
colchicine: DT, drug therapy
cyclosporin: DT, drug therapy
levamisole: DT, drug therapy
mesalazine: DT, drug therapy
pentoxifylline: DT, drug therapy
thalidomide: DT, drug therapy
RN (colchicine) 64-86-8; (**cyclosporin**) 79217-60-0; (levamisole) 14769-73-4, 16595-80-5; (mesalazine) 89-57-6; (pentoxifylline) 6493-05-6; (**thalidomide**) 50-35-1

L14 ANSWER 24 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB Oral manifestations are a common feature of human immunodeficiency virus (HIV) infection. They may present as neoplasms, opportunistic infections, or other lesions. The dermatologist may be the first health care provider to suspect HIV infection when recognizing some of the **oral lesions** described in this article. Some of these lesions may be of prognostic significance for the subsequent development of AIDS. Management of the **oral lesions** can significantly reduce morbidity and improve quality of life.
AN 94282420 EMBASE
DN 1994282420
TI The mouth in human immunodeficiency virus infection.
AU Greenspan D.; Greenspan J.S.
CS Department of Stomatology, UCSF, Box 0422,San Francisco, CA 94143-0422, United States
SO Seminars in Dermatology, (1994) 13/2 (144-150).
ISSN: 0278-145X CODEN: SDERDN
CY United States
DT Journal; General Review
FS 004 Microbiology
011 Otorhinolaryngology
013 Dermatology and Venereology
037 Drug Literature Index
LA English
SL English
SO Seminars in Dermatology, (1994) 13/2 (144-150).
ISSN: 0278-145X CODEN: SDERDN
AB . . . other lesions. The dermatologist may be the first health care provider to suspect HIV infection when recognizing some of the **oral lesions** described in this article. Some of these lesions may be of prognostic significance for the subsequent development of AIDS. Management of the **oral lesions** can significantly reduce morbidity and improve quality of life.
CT Medical Descriptors:
*acquired immune deficiency syndrome: DI, diagnosis
***aphthous ulcer: DT, drug therapy**
***aphthous ulcer: ET, etiology**

***aphthous ulcer: DI, diagnosis**
*human immunodeficiency virus infection: DI, diagnosis
*mouth infection: DT, drug therapy
*mouth infection: DI, diagnosis
*mouth infection: ET, etiology
*salivary gland disease:. . .
therapy
clotrimazole: AD, drug administration
clotrimazole: DT, drug therapy
corticosteroid: AD, drug administration
corticosteroid: DT, drug therapy
coumarin anticoagulant: IT, drug interaction
coumarin anticoagulant: CB, drug combination
cyclosporin a: IT, drug interaction
cyclosporin a: CB, drug combination
digoxin: IT, drug interaction
digoxin: CB, drug combination
fluconazole: DT, drug therapy
fluconazole: IT, drug interaction
fluconazole: CB, drug combination
fluconazole: AD, drug administration
fluocinonide: AD, drug administration
fluocinonide: DT, drug therapy
itraconazole: CB, drug combination
itraconazole: DT, drug therapy
itraconazole: IT, drug interaction
itraconazole: AD, drug administration
ketoconazole: DT, drug therapy
ketoconazole: IT, drug. . . drug combination
phenytoin: IT, drug interaction
povidone iodine: DT, drug therapy
rifampicin: IT, drug interaction
rifampicin: CB, drug combination
terfenadine: IT, drug interaction
terfenadine: CB, drug combination
thalidomide: DT, drug therapy
unclassified drug
RN. . . 82410-32-0; (retinoic acid) 302-79-4; (zidovudine) 30516-87-1; (amoxicillin plus clavulanic acid) 74469-00-4; (chlorhexidine gluconate) 18472-51-0; (clindamycin) 18323-44-9; (clobetasol) 25122-41-2; (clotrimazole) 23593-75-1; (**cyclosporin** a) 59865-13-3, 63798-73-2; (digoxin) 20830-75-5, 57285-89-9; (fluconazole) 86386-73-4; (**fluocinonide**) 356-12-7; (itraconazole) 84625-61-6; (ketoconazole) 65277-42-1; (metronidazole) 39322-38-8, 443-48-1; (mycolog) 53262-75-2; (nystatin) 1400-61-9, 34786-70-4, 62997-67-5; (orabase) 81209-86-1; (phenytoin) 57-41-0, 630-93-3; (povidone iodine) 25655-41-8; (rifampicin) 13292-46-1; (terfenadine) 50679-08-8; (**thalidomide**) 50-35-1

L14 ANSWER 25 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB Behcet's syndrome is a chronic, inflammatory disorder with organic involvement. It is characterized by the presence of recurrent **oral ulcers** joint to genital ulcers, ocular manifestations and other cutaneous lesions. Less frequently may appear arthritis, thrombophlebitis or neurologic and gastrointestinal involvement. Behcet's disease is associated to HLA B5 (Bw51), mainly in endemic areas and in patients with ocular involvement. Immunitary disturbances against herpes virus infection
and streptococcal antigens have been considered as other ethiologic factors. Vasculitis and pustule formation are the main histologic findings

of the cutaneous lesions. Cellular cytotoxicity and immunocomplexes deposition are main mechanisms involved in the pathogenesis of the lesions. Diagnostic criteria require the presence of recurrent **oral ulcers** joint to two or more of the following manifestations: genital ulcers, ocular involvement, other cutaneous lesions or positive pathergy test. Seroids are the treatment of choice, in some cases associated to other immunosuppressors (azathioprine, cyclophosphamide). Cyclosporine A is the treatment of choice for cases with severe ocular involvement and **thalidomide** is a good alternative for steroids in cases with recurrent oral or genital ulcers without systemic involvement.

AN 94193408 EMBASE
DN 1994193408
TI [Behcet disease. Clinical and therapeutical trends].
ENFERMEDAD DE BEHCET. ACTUALIZACION CLINICO-TERAPEUTICA.
AU Sanmartin Jimenez O.; Botella Estrada R.; Vilata Corel J.J.
CS Servicio de Dermatologia, Hospital General Universitario,Valencia, Spain
SO Monografias de Dermatologia, (1994) 7/2 (70-78).
ISSN: 0214-4220 CODEN: MONDE4
CY Spain
DT Journal; Article
FS 013 Dermatology and Venereology
031 Arthritis and Rheumatism
037 Drug Literature Index
LA Spanish
SL Spanish; English
SO Monografias de Dermatologia, (1994) 7/2 (70-78).
ISSN: 0214-4220 CODEN: MONDE4
AB Behcet's syndrome is a chronic, inflammatory disorder with organic involvement. It is characterized by the presence of recurrent **oral ulcers** joint to genital ulcers, ocular manifestations and other cutaneous lesions. Less frequently may appear arthritis, thrombophlebitis or neurologic and gastrointestinal. . . and immunocomplexes deposition are main mechanisms involved in the pathogenesis of the lesions. Diagnostic criteria require the presence of recurrent **oral ulcers** joint to two or more of the following manifestations: genital ulcers, ocular involvement, other cutaneous lesions or positive pathergy test.. . . associated to other immunosuppressors (azathioprine, cyclophosphamide). Cyclosporine A is the treatment of choice for cases with severe ocular involvement and **thalidomide** is a good alternative for steroids in cases with recurrent oral or genital ulcers without systemic involvement.
CT Medical Descriptors:
*behcet . . . EP, epidemiology
article
clinical feature
human
oral drug administration
pathogenesis
prognosis
*azathioprine: DT, drug therapy
*chlorambucil: DT, drug therapy
*colchicine: DT, drug therapy
*corticosteroid: DT, drug therapy
*cyclophosphamide: DT, drug therapy
***cyclosporin: DT, drug therapy**
*dapsone: DT, drug therapy
*prednisone

RN (azathioprine) 446-86-6; (chlorambucil) 305-03-3; (colchicine) 64-86-8; (cyclophosphamide) 50-18-0; (**cyclosporin**) 79217-60-0; (dapsone) 80-08-0; (prednisone) 53-03-2

L14 ANSWER 26 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AB Next to caries and periodontal diseases, recurrent **aphthous** ulceration (RAU) which affects about 20% of the general population is the most common oral disease. Hippocrates named the disease about 400 b.C., and the word **aphthous** (.alpha..pi..tau..epsilon..iota..nu. = to set fire) refers to the typical prodromal burning sensation. With the uncertain etiology, no known laboratory procedures have thus far been of value to confirm the diagnosis which, together with a history of reappearing ulcers, is entirely based on clinical criteria. According to varying clinical characteristics, RAU is classified as minor, major and herpetiform ulcers. Although usually an isolated intraoral disease in otherwise healthy individuals, RAU is also the mainstay symptom in Behcet's syndrome, and the two entities are considered cause-related. A variety of local and systemic factors may provoke RAU recurrences in predisposed individuals. Emotional stress, for instance, usually figures in the row of triggering stimuli, but recent studies have not supported this contention. Hematinic deficiencies, in particular deficits of iron and vitamins B, have also been considered provoking factors by some authors. However, rather than being precipitating circumstances, deficiences may be due to nutrimental waste secondary to an infective process. Previous etiological hypotheses have included autoimmunity, cross-reactions between streptococci and oral mucosal cells, and a viral etiology mainly focusing on HSV and adenoviruses. Supporting evidence for such theories is, however, lacking. The hypothesis of the present review is that RAU is caused by reactivation of a locally latent herpesvirus other than HSV with RAU being a clinical manifestation of the host immune response towards virally infected oral epithelial cells. Clinically, the local, recurrent, self-limiting nature of RAU together with the prodromal burning sensation and increased susceptibility for mechanical trauma as regards the development of ulcers makes the disease very similar to recurrent herpes labialis although the localization of lesions is different. In contrast to recurrent herpes labialis, however, transmission of RAU has never been reported in the literature. Still, a possible transference has been confirmed by some patients. Studies have generally failed to isolate viruses and to detect viral antigens from lesions, and the possible implication of latent viruses has only lately been accounted for. A recent serological study has demonstrated specific VZV IgM antibodies in approximately 50%, and CMV IgM antibodies in about 25% of the patients in association with recurrences, possibly reflecting reactivation of these viruses. However, polyclonal B-cell stimulation due to cross-reactions with other viruses cannot be excluded. By polymerase chain reaction, one part of VZV DNA has been detected in all of the thus far examined ulcers, and a preliminary study has demonstrated CMV DNA in 38% of preulcerative specimens. Although these findings may support an etiological implication of VZV and/or CMV, the presence and possible causal significance of other viral agents cannot be precluded. RAU patients are characterized by systemic and oral mucosal cellular immunosuppression. Systemically, the immunosuppression is typified by a T-cell imbalance with a decreased fraction of CD4+ cells and/or an increased fraction of CD8+ cells. The oral mucosa of RAU-susceptible individuals is characterized by decreased numbers of both CD4+ and CD8+ cells. Whereas the peripheral T-cell imbalance is analogous to the imbalance in many viral infections, little is known about mucosal resistance to viral infections. It is, however, generally accepted that reactivation of latent herpesvirus infections is favoured by impaired

cellular immune defenses, just as herpesvirus infections themselves may induce cellular immunosuppression. Thus, the reduced number of T-lymphocytes in the oral mucosa might either favour reactivation of latent herpesviruses or reflect latency hereof. There is no substantial evidence for non-specific lymphocyte dysfunctions in RAU patients. However, there are indications of some cell-mediated responses being depressed during exacerbations which interestingly may correspond to the anergy observed during some viral infections.

AN 94037193 EMBASE
DN 1994037193
TI Recurrent **aphthous** ulceration: Virological and immunological aspects.
AU Pedersen A.
CS Dept. of Oral Medicine/Oral Surgery, University Hospital,Copenhagen, Denmark
SO APMIS, Supplement, (1993) 101/37 (1-37).
ISSN: 0903-465X CODEN: APSUEN
CY Denmark
DT Journal; General Review
FS 013 Dermatology and Venereology
037 Drug Literature Index
026 Immunology, Serology and Transplantation
LA English
SL English; Danish
TI Recurrent **aphthous** ulceration: Virological and immunological aspects.
SO APMIS, Supplement, (1993) 101/37 (1-37).
ISSN: 0903-465X CODEN: APSUEN
AB Next to caries and periodontal diseases, recurrent **aphthous** ulceration (RAU) which affects about 20% of the general population is the most common oral disease. Hippocrates named the disease about 400 b.C., and the word **aphthous** (.alpha..pi..tau..epsilon..iota..nu. = to set fire) refers to the typical prodromal burning sensation. With the uncertain etiology, no known laboratory procedures. . .
CT Medical Descriptors:
***aphthous ulcer: ET, etiology**
***aphthous ulcer: DT, drug therapy**
*herpes virus
cellular immunity
human
humoral immunity
priority journal
review
aciclovir: DT, drug therapy
cimetidine: DT, drug therapy
colchicine: DT, drug therapy
cyclosporin a: DT, drug therapy
glucocorticoid: DT, drug therapy
interferon: DT, drug therapy
levamisole: DT, drug therapy
longo vital
plant extract: DT, drug therapy
thalidomide: DT, drug therapy
unclassified drug
RN (aciclovir) 59277-89-3; (cimetidine) 51481-61-9, 70059-30-2; (colchicine) 64-86-8; (**cyclosporin** a) 59865-13-3, 63798-73-2; (levamisole) 14769-73-4, 16595-80-5; (**thalidomide**) 50-35-1

L14 ANSWER 27 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 92221722 EMBASE

DN 1992221722
TI [The treatment of aphthosis].
APHTOSES: APPROCHES THERAPEUTIQUES.
AU Jacobs C.; Marot L.
CS Unite de Dermatol. Professionnelle, Clos Chapelle-aux-Champs 30-33,1200 Bruxelles, Belgium
SO Louvain Medical, (1992) 111/6 (351-353).
ISSN: 0024-6956 CODEN: LOMEAL
CY Belgium
DT Journal; Article
FS 004 Microbiology
011 Otorhinolaryngology
013 Dermatology and Venereology
037 Drug Literature Index
LA French
SO Louvain Medical, (1992) 111/6 (351-353).
ISSN: 0024-6956 CODEN: LOMEAL
CT Medical Descriptors:
***aphthous ulcer: DT, drug therapy**
article
human
*aciclovir: DT, drug therapy
*colchicine: DT, drug therapy
***cyclosporin a: DT, drug therapy**
*dapsone: DT, drug therapy
*etretin: DT, drug therapy
*levamisole: DT, drug therapy
*lidocaine: DT, drug therapy
*methisoprinol: DT, drug therapy
*methotrexate: DT, drug therapy
*nystatin: DT, drug therapy
*tetracycline: DT, drug therapy
***thalidomide: DT, drug therapy**
*triamcinolone: DT, drug therapy
*trichloroacetic acid: DT, drug therapy
choline salicylate
RN (aciclovir) 59277-89-3; (colchicine) 64-86-8; (**cyclosporin** a) 59865-13-3, 63798-73-2; (dapsone) 80-08-0; (etretin) 55079-83-9; (levamisole) 14769-73-4, 16595-80-5; (lidocaine) 137-58-6, 24847-67-4, 56934-02-2, 73-78-9; (methisoprinol) 36703-88-5; (methotrexate) 15475-56-6, 59-05-2, 7413-34-5; (nystatin) 1400-61-9, 34786-70-4, 62997-67-5; (tetracycline) 23843-90-5, 60-54-8, 64-75-5; (**thalidomide**) 50-35-1; (triamcinolone) 124-94-7; (trichloroacetic acid) 14357-05-2, 76-03-9; (choline salicylate) 2016-36-6

L14 ANSWER 28 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 93015605 EMBASE
DN 1993015605
TI Behcet's disease.
AU Stratigos A.J.; Laskaris G.; Stratigos J.D.
CS Division of Dermatology, New England Deaconess Hospital, 110 Francis Street,Boston, MA 02215, United States
SO Seminars in Neurology, (1992) 12/4 (346-357).
ISSN: 0271-8235 CODEN: SEMNEP
CY United States
DT Journal; General Review
FS 008 Neurology and Neurosurgery
012 Ophthalmology
013 Dermatology and Venereology
031 Arthritis and Rheumatism

037 Drug Literature Index
LA English
SO Seminars in Neurology, (1992) 12/4 (346-357).
ISSN: 0271-8235 CODEN: SEMNEP
CT Medical Descriptors:
*behcet disease: DT, drug therapy
*behcet disease: EP, epidemiology
*behcet disease: ET, etiology
aphthous ulcer: DT, drug therapy
arthritis: DT, drug therapy
cardiomyopathy
clinical feature
corticosteroid therapy
genital ulcer: DT, drug therapy
geographic distribution
human
laboratory diagnosis
leukocytoclastic vasculitis
neurologic disease: DT, drug therapy
neuropathology
nuclear. . . imaging
prevalence
retinitis: DT, drug therapy
review
sex difference
*azathioprine: DT, drug therapy
*chlorambucil: DT, drug therapy
*colchicine: DT, drug therapy
*corticosteroid: DT, drug therapy
*cyclophosphamide: DT, drug therapy
***cyclosporin: DT, drug therapy**
dapsone: DT, drug therapy
prostacyclin: DT, drug therapy
thalidomide: DT, drug therapy
RN (azathioprine) 446-86-6; (chlorambucil) 305-03-3; (colchicine) 64-86-8; (cyclophosphamide) 50-18-0; (**cyclosporin**) 79217-60-0; (dapsone) 80-08-0; (prostacyclin) 35121-78-9, 61849-14-7; (**thalidomide**) 50-35-1

L14 ANSWER 29 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 91107709 EMBASE
DN 1991107709
TI Diagnosis and treatment of oral aphtous ulcers.
AU Boisnic S.; Tovaru S.
CS Departement de Stomatologie, Medicale du Dr Szpirglas, Groupe Hosp Pitie-Salpetriere, 47-83 Boulevard de l'Hopital,F-75651 Paris Cedex 13, France
SO Annales de Dermatologie et de Venereologie, (1991) 118/1 (53-59).
ISSN: 0151-9638 CODEN: ADVED7
CY France
DT Journal; Article
FS 011 Otorhinolaryngology
013 Dermatology and Venereology
037 Drug Literature Index
LA French
SO Annales de Dermatologie et de Venereologie, (1991) 118/1 (53-59).
ISSN: 0151-9638 CODEN: ADVED7
CT Medical Descriptors:
***aphthous stomatitis: DI, diagnosis**
***aphthous stomatitis: ET, etiology**

***aphthous stomatitis: DT, drug therapy**
*mouth mucosa
article
human
priority journal
*acetylsalicylic acid: DT, drug therapy
*disinfectant agent: DT, drug therapy
*lidocaine: DT, drug therapy
*pyralvex: DT, drug. . . drug therapy
ascorbic acid: DT, drug therapy
betamethasone: DT, drug therapy
borostyrol: DT, drug therapy
chlorhexidine: DT, drug therapy
colchicine: DT, drug therapy
dapsone: DT, drug therapy
fluocinonide: DT, drug therapy
imudon: DT, drug therapy
levamisole: DT, drug therapy
methisoprinol: DT, drug therapy
prednisone: DT, drug therapy
silver nitrate: DT, drug therapy
thalidomide: DT, drug therapy
trichloroacetic acid: DT, drug therapy
unclassified drug
RN. . . 63781-77-1; (lidocaine) 137-58-6, 24847-67-4, 56934-02-2, 73-78-9; (ascorbic acid) 134-03-2, 15421-15-5, 50-81-7; (betamethasone) 378-44-9; (chlorhexidine) 3697-42-5, 55-56-1; (colchicine) 64-86-8; (dapsone) 80-08-0; (**fluocinonide**) 356-12-7; (levamisole) 14769-73-4, 16595-80-5; (methisoprinol) 36703-88-5; (prednisone) 53-03-2; (silver nitrate) 7761-88-8; (**thalidomide**) 50-35-1; (trichloroacetic acid) 14357-05-2, 76-03-9

L14 ANSWER 30 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB A number of diseases can cause recurrent intraoral ulceration. This review
focuses principally on drug management of intraoral ulceration associated with local and systemic conditions most likely to be observed on an outpatient basis by the general practitioner. These consist of recurrent **aphthous** stomatitis, erosive lichen planus, benign mucous membrane pemphigoid (BMMP), erythema multiforme, Behcet's disease, allergic stomatitis and infection. Information is provided on a spectrum of medication found useful in ulcer management, including topical antimicrobial and antifungal agents, topical and systemic corticosteroids,
topical and systemic analgesics, and systemic immunosuppressive and anxiolytic drugs, plus details of dosage, important adverse reactions and interactions. A treatment guide for management of recurrent **aphthae** is presented. The reader is presumed to be familiar with differential diagnosis and the importance of establishing an accurate impression before starting drug therapy.
AN 90051128 EMBASE
DN 1990051128
TI Pharmacological management of recurrent oral mucosal ulceration.
AU Burgess J.A.; Johnson B.D.; Sommers E.
CS Department of Oral Medicine, University of Washington School of Dentistry,
Seattle, WA 98195, United States
SO Drugs, (1990) 39/1 (54-65).
ISSN: 0012-6667 CODEN: DRUGAY
CY New Zealand

DT Journal; General Review
FS 004 Microbiology
011 Otorhinolaryngology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
SO Drugs, (1990) 39/1 (54-65).
ISSN: 0012-6667 CODEN: DRUGAY
AB . . . and systemic conditions most likely to be observed on an outpatient basis by the general practitioner. These consist of recurrent **aphthous** stomatitis, erosive lichen planus, benign mucous membrane pemphigoid (BMMP), erythema multiforme, Behcet's disease, allergic stomatitis and infection. Information is provided. . . immunosuppressive and anxiolytic drugs, plus details of dosage, important adverse reactions and interactions. A treatment guide for management of recurrent **aphthae** is presented. The reader is presumed to be familiar with differential diagnosis and the importance of establishing an accurate impression. . .
CT Medical Descriptors:
*allergy
***aphthous stomatitis**
***aphthous ulcer**
*behcet disease
*erythema multiforme
*infection
*lichen planus
*pemphigoid
*recurrent disease
*ulcer
candidiasis
gastrointestinal disease: SI, side effect
gingivitis
herpes simplex virus 1
liver toxicity: SI, side effect
photosensitivity: SI, side effect
skin disease:. . .
AD, drug administration
*immunosuppressive agent: DT, drug therapy
*nonsteroid antiinflammatory agent: DT, drug therapy
*nonsteroid antiinflammatory agent: AE, adverse drug reaction
acetylsalicylic acid
alprazolam
carbenoxolone
chlorhexidine gluconate
clotrimazole
cromoglycate disodium
cyanocobalamin
deoxycorticosterone
dexamethasone
diazepam
diphenhydramine
estrogen
fluocinonide
folic acid
hydrogen peroxide
ibuprofen
levamisole
lidocaine

lorazepam
magnesium hydroxide
minocycline
silver nitrate
paracetamol
tetracycline
thalidomide
triamcinolone acetonide

RN. . . 23593-75-1; (cromoglycate disodium) 15826-37-6, 16110-51-3, 93356-79-7, 93356-84-4; (cyanocobalamin) 53570-76-6, 68-19-9, 8064-09-3; (deoxycorticosterone) 64-85-7; (dexamethasone) 50-02-2; (diazepam) 439-14-5; (diphenhydramine) 147-24-0, 58-73-1; (**fluocinonide**) 356-12-7; (folic acid) 59-30-3, 6484-89-5; (hydrogen peroxide) 7722-84-1; (ibuprofen) 15687-27-1; (levamisole) 14769-73-4, 16595-80-5; (lidocaine) 137-58-6, 24847-67-4, 56934-02-2, 73-78-9; (lorazepam) 846-49-1; (magnesium hydroxide) 1309-42-8, 1317-43-7; (minocycline) 10118-90-8, 11006-27-2, 13614-98-7; (silver nitrate) 7761-88-8; (paracetamol) 103-90-2; (tetracycline) 23843-90-5, 60-54-8, 64-75-5; (**thalidomide**) 50-35-1; (triamcinolone acetonide) 76-25-5

L14 ANSWER 31 OF 31 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 91033783 EMBASE
DN 1991033783
TI Management of **aphthous**-like ulcers in HIV disease.
AU Epstein J.B.
CS 1651 Third Avenue, New York, NY 10128, United States
SO AIDS Patient Care, (1990) 4/4 (12-13).
ISSN: 0893-5068 CODEN: APACEF
CY United States
DT Journal; Article
FS 011 Otorhinolaryngology
047 Virology
037 Drug Literature Index
LA English
TI Management of **aphthous**-like ulcers in HIV disease.
SO AIDS Patient Care, (1990) 4/4 (12-13).
ISSN: 0893-5068 CODEN: APACEF
CT Medical Descriptors:
***aphthous ulcer: DT, drug therapy**
*human immunodeficiency virus
article
case report
human
oral drug administration
topical drug administration
***fluocinonide: DT, drug therapy**
*methylprednisolone sodium succinate: DT, drug therapy
*methylprednisolone sodium succinate: CB, drug combination
*prednisone: DT, drug therapy
***thalidomide: DT, drug therapy**
oxycodone: DT, drug therapy
oxycodone: CB, drug combination
RN (**fluocinonide**) 356-12-7; (methylprednisolone sodium succinate) 2375-03-3, 2921-57-5; (prednisone) 53-03-2; (**thalidomide**) 50-35-1; (oxycodone) 124-90-3, 76-42-6

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L14 ANSWER 7 OF 31 USPATFULL
AB Methods of treatment for inflammatory and autoimmune dermatoses which comprises topical and/or systemic administration of a therapeutically-effective amount of **thalidomide** alone or in combination with other dermatological agents.
AN 97:68480 USPATFULL
TI Treatment of inflammatory and/or autoimmune dermatoses with **thalidomide** alone or in combination with other agents
IN Andrulis, Jr., Peter J., Bethesda, MD, United States
Drulak, Murray W., Gaithersburg, MD, United States
PA Andrulis Pharmaceuticals, Beltsville, MD, United States (U.S. corporation)
PI US 5654312 19970805 <--
AI US 1995-475426 19950607 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Nutter, Nathan M.
LREP Angres, Isaac
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 925
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Treatment of inflammatory and/or autoimmune dermatoses with **thalidomide** alone or in combination with other agents
PI US 5654312 19970805 <--
AB Methods of treatment for inflammatory and autoimmune dermatoses which comprises topical and/or systemic administration of a therapeutically-effective amount of **thalidomide** alone or in combination with other dermatological agents.
SUMM The present invention relates to novel methods for treating inflammatory and/or autoimmune dermatoses with **thalidomide** alone or in combination with other agents. The present invention also relates to methods of treating dermatoses with inhibitors of. . .
SUMM . . . be triggered by a number of external events ranging from exposure to UV light from the sun to an allergen. **Thalidomide** has been demonstrated to have an inhibitory effect on the pro-inflammatory cytokines. It has been shown to inhibit TNF-alpha production. . . stimulated monocytes (Sampaio et al., J. Exp. Med., 173:699-703, 1991). Moreira et al. (J. Exp. Med., 177:1675-80, 1993) reported that **thalidomide** acts by enhancing TNF-alpha m-RNA degradation. Shannon et al. (Amer. Society for Microbiology Ann. Mtg., Abs. U-53, 1990) indicated **thalidomide** inhibited IL-1 beta production in vitro. Such an inhibitory effect on IL-1 beta may be direct or indirect through TNF-alpha. . .
SUMM . . . surface of endothelial cells facilitates the binding of inflammatory cells that is a precondition to transendothelial migration occurring during inflammation. **Thalidomide** also has an anti-angiogenic effect since TNF-alpha stimulates endothelial cell motility in vitro (Leibovich, Nature, 329:630-32, 1987; Rosen et al.,. . . et al., Proc. Natl. Acad. Sci. (USA), 84:5277-5291, 1987). D'Amato et al. (Proc. Natl. Acad. Sci. (USA), 91:4082-5,1994) showed that **thalidomide** was an effective inhibitor of angiogenesis induced by bFGF.
SUMM In 1965 Sheskin (Lepr. Rev., 36:183-7) administered **thalidomide** to leprosy patients suffering from the complication erythema nodosum leprosum (ENL), to sedate them. ENL is characterized by recurrent erythematosus nodules on the skin, weight loss, mania, neuritis, fever,

malaise, and sometimes epididyo-orchitis. Within 12 hours of **thalidomide** administration nodular eruptions began to heal and within two days fever declined and the ENL lesions had completely resolved. In. . . double blind clinical trial conducted in four countries and coordinated by the World Health Organization, which tested
the efficacy of **thalidomide** versus aspirin for treatment of ENL. The conclusions reached supported Sheskin's original observations about the effectiveness of **thalidomide** for treatment of ENL. Wemambu et al. (Lancet, 2:933-5, 1969) observed necrotizing vasculitis of veins and arteries in patients with. . . Appl. Immun., 57:317-332 (1978) showed in a study of neutrophil activation in ENL patients just before and during treatment with **thalidomide** that tissue damage was not due solely to neutrophil activation as occurs in immune complex diseases, but rather neutrophils appeared to be activated by an undefined lymphokine. This group went on to state that the therapeutic effect of **thalidomide** was not due to inhibition of neutrophil activation. Sarno et al. (Clin. Exp. Immunol., 84:103-8, 1991) showed that TNF-alpha levels were elevated in ENL patients and that TNF-alpha had a major role in the pathogenesis of this disease. **Thalidomide** was shown to inhibit TNF-alpha production in these ENL patients. Sampaio et al. (J. Inf. Dis., 168:408-14, 1993) confirmed Sarno's. . .

SUMM The fortuitous finding that **thalidomide** was effective in treating ENL stimulated other investigators to look at the efficacy of **thalidomide** for treating other dermatoses with a possible inflammatory and/or autoimmune pathogenesis.

SUMM . . . areas of the body. Its etiology is unknown. Londono (Int. J. Dermatol., 12:326-8, 1973) was the first to report using **thalidomide** as a treatment for actinic prurigo. He administered 300 mg of **thalidomide** per day to 34 patients until clinical improvement was noted and then reduced the dosage progressively. There was notable improvement. . . an immunological etiology. Lovell et al.
(Brit. J. Dermatol, 108:467-71, 1983) treated 14 actinic prurigo patients with 50-100 mg of **thalidomide** per day for children and 100-200 mg of **thalidomide** per day for adults, for variable periods of time. Eleven patients had long term clinical improvement and three were free of symptoms even after **thalidomide** was discontinued. No side effects were noted.

SUMM . . . on the basis of clinical criteria. Mattos (Bol. Div. Nac. Lepra., 32:71) in 1973 was the first investigator to use **thalidomide** to treat prurigo nodularis. One of the two patients treated received 200 mg per day of **thalidomide** and the other patient, a woman, received 300 mg daily. Both patients had excellent clinical responses to the therapy after several weeks. Sheskin (Hautarzt, 26:215, 1975) reported treating three prurigo nodularis patients with **thalidomide**. These patients suffered from the disease for eight to twenty-four years, but responded clinically within a few weeks of initiation of **thalidomide** therapy. Other studies (Van den Broek, Arch. Dermatol, 116:571, 1980; Nikolowski, Hautarzt, 31:565, 1980; Winkelmann et al., Acta. Dermato-Venereologica, 64:412-7,. . . the intensive itch that accompanies this condition subsiding within 2-3 weeks of the start of 200 mg per day of **thalidomide**. However, in these studies it was noted that it takes at least six months of **thalidomide** therapy before strongly lichenified lesions completely heal.

SUMM . . . certain drugs. Barba-Rubio and Gonzalez, Derm. Rev. Mex., 19:131 (1975) treated 20 discold lupus erythematosus patients with 300 mg of **thalidomide** per day. Within two weeks 19 of these

patients responded clinically and the medication was then reduced to a maintenance. . . al., Giorn. Ital. Derre. Vener, 115:471, 1980; Samsoen et al., Ann. Dermatol Venereol (Paris), 107:515-23, 1980) confirmed the effectiveness of **thalidomide** therapy in treating discold lupus erythematosus patients refractory to other treatments such as steroids. In most instances a clinical effect was detected within 14 days of initiation of 100-200 mg per day of **thalidomide**, however, a total and definite recovery was seen in only 15-20% of patients. In most patients a 25-50 mg per day maintenance dose of **thalidomide** was required to sustain a clinical improvement.

SUMM **Thalidomide** has also been used successfully to treat Behcet's syndrome, a rare and severe illness of unknown etiology often afflicting young. . . and genitalia, uveitis, and retinal vasculitis. There also may be atrophy of the gastrointestinal tract and pulmonary or myocardial fibrosis. **Thalidomide** therapy was an important breakthrough, because prior to this there was no specific treatment for Behcet's syndrome. Steroids proved to. . . prescribed (Mamo et al., Arch. Ophthamol, 71:4-14, 1964). Saylan and Saltik (Arch. Dermatol, 118:536, 1982) were the first to use **thalidomide** to treat 22 patients with Behcet's syndrome who had deep and persistent oral **aphthae** . Patients were initially administered 400 mg per day of **thalidomide** for five days followed by 200 mg per day for 15 to 60 days. This regimen resulted in rapid and complete healing of **aphthae**. Torras et al. (Arch. Dermatol, 118:875, 1982) found that there was complete healing of giant **aphthae** in eight of nine Behcet's patients treated with 100 mg per day of **thalidomide** for 10 days. Jorizzo et al. (Arch. Int. Med., 146:878-81, 1986) reported similar success with **thalidomide** in five patients with Behcet's syndrome. In 1993 Denman et al., Rev. Med. Int., 14:(suppl 1) 495, treated 39 patients with Behcet's syndrome with 50 mg of **thalidomide** three nights per week for a mean time of 35.9 months and a maximum treatment time of up to 65 months. Concomitant treatment in this patient group included 10 patients on prednisone, 3 on azathioprine and 1 patient on **cyclosporin**. Mucosal lesions healed in all patients. Moulin et al. (Ann. Dermatol Venereol, 110:611, 1983) used 100 mg per day of **thalidomide** to treat six patients with a Jessner-Kanof lymphocytic infiltration of the skin. This disease is characterized by numerous lesions on. . . i:251 (1977) treated a patient with a relapsing non-suppurative panniculitis termed Weber Christian Disease, with 300 mg per day of **thalidomide** for three weeks which was reduced to 200 mg per day and then to 100 mg per day after 10. . . lesions steadily regressed during therapy and it was reported that a disease free state was maintained for 13 months after **thalidomide** was stopped. **Thalidomide** has also been used to treat recurrent erythema multiforme, a flu like syndrome in which blisters appear on mucous membranes. . . Bahmer et al., Acta. Derm. Venereal, 62:449 (1982) treated a patient who had recurrent erythema multiforme with 200 mg of **thalidomide** per day. Within a few days the mucosal membrane and skin lesions healed and the daily dosage of **thalidomide** was lowered. The patient has been maintained in a disease free state by administration of 100 mg of **thalidomide** per day.

SUMM As indicated oral administration of **thalidomide** has been

successfully used to treat a limited number of dermatoses that may have an autoimmune and/or inflammatory component associated with them. Topical application of **thalidomide** is a useful therapeutic approach for disease states with an autoimmune and/or inflammatory basis. Furthermore, **thalidomide** may be used alone to treat dermatoses with an autoimmune and/or inflammatory basis or in unique combinations with other cytokine/growth. . . anti-inflammatory and/or

anti auto-immune agents and/or other physical and/or chemical dermatological treatments. An example of such combination therapy could involve **thalidomide** given with pentoxifylline and a glucocorticoid such as dexamethasone. The activity of each of these agents would be expected to. . . a different point in this synthesis.

Pentoxifylline inhibits TNF-alpha gene transcription (Doherty et al., Surgery (St. Louis), 110:192, 1991), while **thalidomide** enhances TNF-alpha m-RNA degradation (Moreira et al., 1993) and glucocorticoids such as dexamethasone inhibit TNF-alpha m-RNA translation (Han et al.,. . .

SUMM **Thalidomide** has been administered orally, however, it may be used topically to treat dermatoses with an autoimmune and/or inflammatory component associated. . .

SUMM **Thalidomide** was first synthesized and marketed in the 1950's as a sedative. The toxicity of the compound was so low that a dose killing 50% of animals (LD.sub.50) could not be established. **Thalidomide** was therefore thought to be a safer alternative to barbiturates. In 1961 **thalidomide** administered to pregnant women resulted in an epidemic of congenital malformation. The incidence of malformed babies paralleled the sales of **thalidomide** and quickly dropped off when **thalidomide** was removed from the market.

SUMM Oral administration of **thalidomide** in the range of 100-200 mg in adult humans results in a peak blood level of 0.9-1.5 mg/liter after 4-6 hours. Hydrolytic cleavage of **thalidomide** occurs in vitro, the rate of which increases as the pH increases. However, hydrolytic cleavage of **thalidomide** in serum is much slower than in vitro at pH 7.4. This may be due to **thalidomide** being highly bound to plasma proteins. Studies in animals demonstrated high **thalidomide** concentrations in the gastrointestinal tract, liver and kidneys with lower concentrations in muscle, brain and adipose tissue. In pregnant animals, **thalidomide** can pass across the placenta.

SUMM Although a complete study of **thalidomide** metabolism in humans has not been performed, in animals the main pathway for **thalidomide** breakdown appears to be nonenzymatic hydrolytic cleavage. Even though immunomodulatory effects of **thalidomide** have not been clearly defined at the molecular level, **thalidomide** has been used to treat the following immunologically-based diseases: acute **aphthous** ulcers (Jenkins et al., Lancet, 2:1424-6, 1984; Grinspan, J. Amer. Acad. Dermatol, 12:85-90, 1985; Revuz et al., Arch. Dermatol, 126:923-7,. . . J., 1:792, 1979) and discold lupus erythematosus (Knop et al., Arch. Dermatol Res., 271:165-70, 1981). In these studies, dosages of **thalidomide** ranging from 100 mg/day to 800 mg/day were administered without serious side effects.

SUMM A further objective of the present invention is the treatment of dermatoses with an autoimmune and/or inflammatory component with **thalidomide** alone or in combination with other agents that inhibit cytokines and/or growth factors, and/or with other classes of therapeutics used. . .

SUMM Another objective of the present invention is the use of **thalidomide** alone or in combination with other agents.

SUMM . . . objective of the current invention is to provide a method for treating dermatoses with an autoimmune and/or inflammatory component with **thalidomide** at a given regimen.

SUMM A further objective of the present invention is a method for the treatment of dermatoses which comprises therapy with **thalidomide** and other drugs on alternative days by diverse schedules.

SUMM An additional objective of the current invention is to utilize **thalidomide** alone or in combination with other inhibitors of cytokines and/or growth factors and/or other treatments for dermatoses as a maintenance. . .

SUMM A still further objective of this invention is to use **thalidomide** alone or in combination with other inhibitors of cytokines and/or growth factors and/or other treatments for dermatoses as a prophylactic. . .

SUMM . . . dermatoses in a mammal which comprises applying and/or administering to said mammal a composition comprising: (a) an effective amount of **thalidomide** and (b) a therapeutically-acceptable vehicle for the **thalidomide**.

SUMM . . . selected from the group consisting of TNF-alpha inhibitors, basic fibroblast growth factor inhibitors and IL-1 beta inhibitors. Typical inhibitors include **thalidomide** and pentoxifylline but the invention is not limited to those.

SUMM The following is a list of examples of dermatological conditions for which **thalidomide** therapy as proposed in this application is useful. However, proposed **thalidomide** treatments will not be limited to these indications since there may be other dermatological conditions not mentioned here where **thalidomide** may also be effective as a therapeutic:

SUMM (r) Diseases of Mucous Membranes: such as **aphthous** ulcers.

SUMM In treating Kaposi's Sarcoma, an ointment containing 10% by weight of **thalidomide** is applied to the lesion. In an alternative embodiment, Kaposi's Sarcoma is treated concurrently by topical and oral treatment. For. . . presenting with Kaposi's Sarcoma is treated daily for two to four weeks with a dosage amount of 50 mg of **thalidomide** a day while an ointment containing 10% by weight **thalidomide** is applied to the lesion three times a day for two to four weeks.

SUMM When used alone, the topically effective amounts of **thalidomide** are typically 5 to 15% by weight in an ointment and is applied one to three times a day for. . .

SUMM Under certain circumstances, it is desirable to administer **thalidomide** therapy simultaneously with other dermatological active agents. For example, a cream containing 5% by weight of **thalidomide** can be administered three times a day while the patient is being given a topical treatment with 1% hydrocortisone. Concurrent administration of oral **thalidomide** with topical **thalidomide** is also a desirable therapeutic goal.

SUMM Additionally, applicants propose to use **thalidomide** alone or in combination with other inhibitors of cytokines and/or growth factors to treat dermatoses. An example of such a combination therapy utilizes **thalidomide** given with pentoxifylline and a glucocorticoid such as dexamethasone. The activity of each of these agents would be expected to. . . these agents acts as an inhibitor at a different point in this synthesis. Pentoxifylline inhibits TNF alpha gene transcription, while **thalidomide** enhances TNF alpha m-RNA degradation and

glucocorticoids, such as dexamethasone, inhibit TNF alpha m-RNA translation.

SUMM The precise amount of **thalidomide** used alone or with other dermatologic agents varies depending, for example, on the condition for which the drug is administered and the size and kind of the mammal. Generally speaking the **thalidomide** can be employed in any amount effective in the treatment of dermatoses.

SUMM For humans, typically-effective amounts of **thalidomide** for use in the topical dosage forms compositions of the present invention range from 5-15% by weight active, however, greater. . .

SUMM . . . be obvious to those skilled in the art that the following dosage forms may comprise as the active component either **thalidomide** alone or in combination with other compounds. Preferably the compounds of the present invention are administered orally, intramuscularly, topically, subcutaneously,. . .

SUMM It is also possible to administer **thalidomide** in a time-release formulation. A wide variety of methods are now available in the art for preparing time-release or long-acting. . . suitable in the practice of the present invention as long as it does not adversely affect the effectiveness of the **thalidomide** in the treatment of dermatoses. Advantages of time-release formulations include a lower concentration of peak serum absorption which substantially reduces. . . A frequency of administration of every 12 or 24 hours would be preferred. In addition, more constant serum concentration of **thalidomide** would result thereby allowing a more consistent relief of symptoms.

DETD

	Quantity (mg/capsules)
Thalidomide	250
Starch dried	200
Magnesium stearate	10

DETD

	Quantity (mg/tablet)
Thalidomide	250
Cellulose, microcrystalline	400
Silicon dioxide, fumed	10
Stearic acid	5

DETD

Thalidomide	60	mg
Starch	45	mg
Microcrystalline cellulose	35	mg
Polyvinylpyrrolidone (as 10% solution in water)	4	mg
Sodium carboxymethyl starch	4.5	mg
Magnesium stearate	0.5	mg
Talc. . .		

DETD

Thalidomide	80	mg
Starch	59	mg

Microcrystalline cellulose	59	mg
Magnesium stearate	2	mg
Total	200	mg

DETD

Thalidomide	150	mg
Starch	164	mg
Microcrystalline cellulose	164	mg
Magnesium stearate	22	mg
Total	500	mg

DETD A topical ointment containing **thalidomide** is prepared as follows:

DETD

	% by weight
Thalidomide	20%
Vegetable oil	10%
Acetyl lanolin	10%
Lanolin alcohol	12%
Sorbitol sesquioleate	20%
Water add to	100%

DETD

	% by weight
Thalidomide	15%
Carboxyvinyl polymers	2%
Preservative	0.01%
Water add to	100%

DETD

Thalidomide	6.0	g
Stearyl alcohol	3.0	g
Lanolin	5.0	g
Vaseline	15.0	g
d H.sub.2 O added to	100.0	g

DETD Liposomes containing **thalidomide** are made as follows:

DETD Ointment containing **thalidomide:**

DETD

Thalidomide	0.9	g
Hydrocortisone	0.1	g
Isopropyl myristate	81.7	g
Liquid petrolatum oil	9.1	g
Silica - aerosil 200	9.18	g

DETD Twenty patients suffering from psoriasis are to be treated with a cream containing 8% by weight of **thalidomide.**

DETD . . . commercially available product. This commercially available product should be designated the "control", whereas the cream containing 8% by weight of **thalidomide** should be the "test" cream.

DETD These data will clearly demonstrate that the therapeutic composition according to the invention containing 8% by weight **thalidomide**

is efficacious and, furthermore, is preferred by the patient to a widely used commercially-available pharmaceutical preparation.

DETD Forty patients suffering from moderate acne are to be treated with a cream containing 5% by weight **thalidomide**.

DETD . . . of the pharmaceutical composition according to the invention, the clinical study should compare this composition with an appropriate placebo (without **thalidomide**) and another commercially available product specifically prescribed for the treatment of acne.

DETD Upon completion of the treatment period, the areas treated with the 5% by weight **thalidomide** cream will exhibit a clinically significant decrease in the severity of acne as compared to placebo treatment. Furthermore, the **thalidomide**-treated subjects will exhibit less severe side effects and complaints as compared to some other commercially available treatments.

DETD . . . exhibiting leg lesions and diagnosed as being Kaposi's sarcoma are to be treated with a cream containing 10% by weight **thalidomide**.

DETD . . . of the pharmaceutical composition according to the invention, the clinical study should compare this composition with an appropriate placebo (without **thalidomide**) and another commercially available product specifically prescribed for the treatment of Kaposi's sarcoma.

DETD . . . Example 13, two patients are treated except that concurrently with topical administration they are orally treated with 50 mg/day of **thalidomide** for the duration of the topical treatment.

CLM What is claimed is:

. . . mammal which comprises administering to said mammal a therapeutically effective amount of a composition comprising: (a) an effective amount of **thalidomide** and (b) a therapeutically acceptable vehicle for **thalidomide**.

12. The method of claim 11 wherein said TNF alpha inhibitor is selected from the group consisting of **thalidomide** and pentoxifylline.

. . . applying to involved areas of the body and/or administering to said mammal a composition comprising: (a) an effective amount of **thalidomide** and; (b) a therapeutically-acceptable vehicle for the **thalidomide**.

. . . 14. A dermatological composition suitable for treating inflammatory and autoimmune dermatoses in a mammal comprising: a) an effective amount of **thalidomide**; (b) an effective amount of an addition dermatologic drug selected from one group consisting of menthol, phenol, camphor, coal tar. . .

IT 50-23-7, Hydrocortisone **50-35-1**, Thalidomide 53-06-5, Cortisone 57-62-5, Aureomycin 69-72-7, Salicylic acid, biological studies 76-22-2, Camphor 89-78-1, Menthol 108-46-3, Resorcinol, biological studies 108-95-2, Phenol, biological studies 130-26-7, Vioform 1314-13-2, Zinc oxide, biological studies 1404-04-2, Neomycin 1405-41-0, Garamycin **6493-05-6** 7439-97-6D, Mercury, ammoniated, biological studies 7704-34-9, Sulfur, biological studies 65454-29-7, Chloromycin
(pharmaceutical compns. contg. thalidomide for treatment of inflammatory and/or autoimmune dermatoses)